In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 21-1513V

PORTIA EXUM,	*	Chief Special Master Corcoran
Petitioner,	*	Dated: May 27, 2025
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Amber Diane Wilson, Wilson Science Law, Washington, DC, for Petitioner.

Mary Novakovic, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION ON REMAND[1]

On June 25, 2021, Portia Exum filed a petition seeking compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petitioner alleges that the tetanus-diphtheria-acellular pertussis ("Tdap") and measles-mumps-rubella ("MMR") vaccines she received on August 20, 2018, caused her to develop autoimmune hepatitis ("AIH"). Pet. at 1.

A one-day Entitlement Hearing was held on March 7, 2024, and after listening to the witnesses' testimony and evaluating the record, I determined that Petitioner was not entitled to compensation. However, Petitioner prevailed on a motion for review, and the Court has ordered me on remand to more fully describe the rationale for my conclusions (including my decisions to accept, reject, and/or credit certain medical literature and expert testimony), and to revise my analysis pertaining to alternative causes. Remand Order, dated Feb. 26, 2025 (ECF No. 82)

[1] Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Ruling will be available to the public in its present form. *Id.*

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) ("Vaccine Act" or "the Act"). Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

("Remand Order") at 44, 50–51. I have now done so—but I reach the same conclusion that I did after hearing. For, as discussed below, Petitioner was unable to preponderantly establish that the Tdap and MMR vaccines can cause AIH, or did so in her case.

I. Factual Background

Pre-Vaccination History

Ms. Exum was born on January 29, 1988. Prior to the vaccinations at issue, she had a history of gastrointestinal reflux issues, small intestinal bacterial overgrowth, and kidney stones. Ex. 2 at 9–12; Ex. 3 at 273–75, 265–71. Notably, during a May 2018 ER visit for treatment of kidney stones, Petitioner's AST and ALT levels (liver enzymes) were tested but found to be normal. Ex. 3 at 268. She also reported to an endocrinologist (who she saw in July 2018 for follow-up regarding her kidney stone issues) that she had been taking certain mushrooms as an immune booster, in anticipation of a trip she planned for later that year. Ex. 1 at 14.

Petitioner began preparing for overseas travel to Kenya and Tanzania in mid-August 2018. *See* Declaration, dated June 8, 2021, filed as Ex. 11 (ECF No. 7-2) ("Exum Decl.") at 1 ¶7. As part of that preparation, she received anti-malarial medication on August 17, 2018. Ex. 3 at 74. She was instructed to begin taking the medication two days before visiting areas with high risk for malaria, and to continue taking it for seven more days after leaving. *Id*. Three days later, on August 20, 2018, as additional preparation for her trip, she received the MMR and Tdap vaccines from her employer's health clinic, but declined the typhoid vaccine. *Id.* at 72–73; Exum Decl. at 1 ¶7. Petitioner also at this time was provided traveler's advisory information about risks of mosquito-borne illnesses, diarrhea, and "malaria prophylaxis." Ex. 3 at 72.

There is no medical record evidence that Petitioner experienced any immediate reaction to either of the vaccines she received on August 20th, or any notable symptoms in the more than one-week period before travelling abroad.

Post-Vaccination Period and Symptoms Onset

Petitioner traveled to Kenya and Tanzania as planned, from August 29 to September 8, 2018. Ex. 4 at 35; Exum Decl. at 1 ¶8. While abroad, she reports having received four or five bug bites. Ex. 4 at 35. Upon return, she felt fatigued, and had symptoms of gastroesophageal reflux disease ("GERD") and indigestion in late September. *Id.*; Ex. 4 at 8. There is no other record evidence from the month of September suggesting Petitioner was experiencing unusual inflammation or signs of an infectious process.

Petitioner has alleged that she began to experience daily nausea in October 2018. Ex. 4 at 35; Exum Decl. at 1 ¶¶10–11. But there is no record evidence she sought treatment for it at this time. At most, in a medical encounter in January 2019, Petitioner stated that her nausea felt especially strong after a workout in mid-October. Ex. 4 at 8.

Later that same month, on October 26, 2018 (now over two months since the vaccinations at issue—and six weeks after return from travel), Petitioner had a routine physical for life insurance purposes. The record from this visit memorializes no complaints or reports of gastrointestinal concerns, fatigue, or any other clinical symptoms. However, testing performed at this time revealed the presence of elevated liver enzymes. Ex. 4 at 42 (ALT of 818 U/L with a 0-45 U/L normal range, AST of 546 U/L with a 0-33 U/L normal range). She did not at this time, however, test positive for biomarkers supporting the presence of inflammation, like "BUN" or creatinine,[3] and she tested negative for Hepatitis C antibody (which would have suggested the presence of an acute or chronic infection that could result in liver disease). *Id.* at 40–41.

Petitioner followed up with a gastroenterologist a month later, on November 28, 2018, to address both the elevated liver enzymes and her ongoing nausea, as well as related GI symptoms. Ex. 3 at 280. An abdominal exam was unremarkable, with no signs of liver enlargement or tenderness. *Id.* A physician's assistant ("PA") noted her recent abnormal liver function tests, however, and that Petitioner reported right-sided distress. *Id.* at 282. The PA recommended testing for an *H. pylori* bacterial infection, and that Petitioner try an over-the-counter anti-acid medication, plus diet modifications to ease her GERD and related symptoms. *Id.* Petitioner was also referred to a hepatologist to have an MRI of her liver. *Id.* at 283. Test results two days after this visit showed even higher AST and ALT levels, but yielded negative results for *H. pylori*. *Id.* at 67–69.

Petitioner's next treatment event occurred over five weeks later, at a visit to her primary care physician ("PCP") on December 7, 2018. Ex. 3 at 61. She now reported upper right quadrant abdominal pain, nausea, fatigue, and yellow eyes. *Id.* An abdominal exam was unremarkable, and her PCP referred her to a hepatologist. *Id*. at 63. She also had her inter-uterine device ("IUD") removed on December 6, 2018, to eliminate it as a potential source for her liver-related issues. *Id.* at 242–43. She then visited the same PCP on December 14, 2018. *Id.* at 57. She now reported that she had not completed the antimalarial drug course prescribed for her, and that some "doctor

[3] The BUN (or "blood urea nitrogen") test is used to measure the amount of urea nitrogen in the blood. *See Blood Urea Nitrogen (BUN) Test,* Mayo Clinic, https://www.mayoclinic.org/tests-procedures/blood-ureanitrogen/about/pac-20384821 (last visited on May 27, 2025). Urea nitrogen is a chemical waste product usually removed from the body through the kidney, so a higher-than-normal BUN test result can stand as evidence that the kidneys or liver may not be working properly. *Id.* Creatinine is a chemical waste product produced by muscle metabolism, and also filtered out by the kidneys (and thus a high reading is further proof of kidney issues). *See* Creatine Test, Mayo Clinic, https://www.mayoclinic.org/tests-procedures/creatinine-test/about/pac-20384646 (last visited on May 27, 2025). An elevated BUN to creatinine measurement is evidence of blood volume depletion. *McKown v. Sec. of Health & Human Services,* No. 15-1451V, 2019 WL 4072113, at *15 (Fed. Cl. Spec. Mstr. July 15, 2019).

friends" of hers had expressed informal views that her condition could be the product of malaria or other insect diseases resulting from big bites she had received while in Tanzania. *Id.*

The PCP ordered additional lab work, and referred her to an infectious disease specialist. Ex. 3 at 59. The lab work again showed elevated AST and ALT, but autoantibody testing for biomarkers associated with AIH (anti-nuclear antibodies and anti-smooth muscle antibodies) were negative. *Id*. at 58.

On December 19, 2018, Petitioner saw hepatologist Dr. Omobonike Oloruntoba for evaluation of her elevated liver enzymes. Ex. 3 at 232–38. Petitioner denied the presence of known risk factors for liver disease, such as alcohol consumption or IV drug use, as well as any medicines or supplements (with the exception of "starting Reishi mushrooms" at some unspecified time). *Id*. at 232. She did, however, acknowledge taking antimalarial medication for ten days in connection with her oversees travel in August-September. *Id*.

Dr. Oloruntoba noted that Petitioner displayed no signs of decompensated liver disease, including icterus, jaundice, confusion, melena, hematochezia, hematemesis, bruising, weight loss, or abdominal swelling, and an abdominal exam was again unremarkable. Ex. 3 at 232–33, 235. But a liver MRI revealed the presence of two hyper-intense lesions consistent with adenomas versus focal nodular hyperplasia ("FNH"), and asymmetric dilation of the left renal vein. *Id*. at 235–36. The diagnostic differential offered by Dr. Oloruntoba included "persistently elevated" liver function tests ("LFTs"), but with a "negative serologic work up," deeming lab results to be "fortunately . . . not consistent with acute liver failure," and hepatic adenoma versus FNH. *Id*. at 237. Additional lab results again showed elevated LFTs, but no signs of an active hepatitis infection. Ex. 8 at 73. Dr. Oloruntoba ordered a liver biopsy and a repeat liver MRI with contrast to be performed in six months. Ex. 3 at 237.

Subsequent Treatment for Hepatitis

Petitioner underwent the liver biopsy on January 3, 2019. Ex. 3 at 227–31. Results established the presence of "marked chronic inflammation," as well as "patchy moderate interface necrosis." *Id.* at 229. The findings were deemed by treaters to support a "broad differential diagnosis" that included "infection, the effects of medications/drugs/herbal remedies, Wilson disease,[4] and autoimmune hepatitis." Ex. 3 at 229.

[4] Wilson disease is a rare, inherited disorder in which the body fails to eliminate excess copper, leading to its accumulation in organs such as the liver or eyes. *Wilson Disease*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=70938&searchterm=Wilson+disease (last visited on May 27, 2025). The build-up of copper can cause significant organ damage and various symptoms, including liver problems. *Id.*

The next day, Petitioner saw hematology and oncology specialist Dr. Charles Eisenbeis, who prepared a write-up/summary of his views on January 12, 2019. Ex. 3 at 221–26. The medical history obtained at this time was consistent with what is described above—that testing had begun to reveal abnormal LFTs in October 2018 and remained elevated thereafter, along with the liver MRI findings. *Id.* at 223. Dr. Eisenbeis also noted, however, that Ms. Exum had experienced no more than mild fatigue as a clinical symptom, and that she had not tested positive for the presence of hepatitis B or C. *Id.* And his own physical exam revealed nothing but the prior-reported nausea plus dyspepsia. *Id.* at 225.

Dr. Eisenbeis observed elevated serum ferritin (iron) levels, which he attributed to "obvious liver disease" (adding that testing for a hereditary condition that could cause excess iron deposits was negative). Ex. 3 at 222. He also suggested Petitioner was experiencing hepatitis, but noted that "[w]orkup so far has been unrevealing for a cause of her liver disease." *Id.*

In mid-January, Petitioner received additional treatment for kidney stones and the lower quadrant pain/nausea she had previously reported. Ex. 2 at 28–30. Labs from this visit also revealed "very elevated" ALT and AST levels. *Id*. at 28. Petitioner also purports to have received an AIH diagnosis around this time frame—although no direct record proof has been offered in which Dr. Oloruntoba, or any other qualified hepatologist, is memorialized to have offered this diagnosis.

On January 29, 2019, petitioner saw an infectious disease specialist, Henry Wu, M.D., for a second opinion regarding the purported AIH diagnosis she had just received. Ex. 4 at 6. At this time, her international travel from the early fall of 2018 was discussed, and she reported that she had entered bodies of water ("she did swim in the Indian Ocean and was in a bay to wade in the water to access the boats"), received several insect bites, and had felt extreme fatigue upon return. *Id*. at 8. Dr. Wu noted that Petitioner was taking a four-to-six-week course of prednisone. *Id*. at 6. He affirmed Petitioner's hepatitis diagnosis (deeming it more likely chronic than attributable to an acute infection) and ordered lab work. *Id.* at 8. The results revealed Petitioner had experienced an Epstein-Barr viral infection at some prior point. *Id*. at 12; Ex. 3 at 207. Her LFTs had also improved but were still elevated. Ex. 3 at 204. Otherwise, no infectious explanation for Petitioner's AIH could be identified. *Id.* at 7.

Petitioner's LFT levels thereafter trended downwards during February and March 2019. Ex. 3 at 43–51. During a GI visit for reflux management in February, Petitioner's treater noted that she was taking kidney-oriented medication in addition to the prednisone to treat her liver issues. *Id.* at 197. Her PCP later noted in April 2019 that her AIH was "improving." *Id*. at 36–37. A visit to a hepatologist that same month revealed continued LFT improvement, although levels remained above normal. *Id.* at 192–96. She also continued to report some ongoing fatigue. *Id.* at 34.

Petitioner visited her hepatologist again in August 2019. Ex. 3 at 162–65. Her LFTs remained elevated, and the hepatologist ordered a metabolic screen to rule out hepatoxicity, and instructed her to continue with her previously-prescribed medications. *Id*. at 165. Labs taken shortly thereafter in September 2019 showed slightly elevated LFTs, but otherwise normal results. *Id.* at 20–31.

By the first half of 2020, Petitioner's liver concerns had mostly resolved, and testing from this time to 2022 revealed normal LFTs. Ex. 10 at 56–61; Ex. 3 at 154. A repeat liver biopsy performed in February 2021, however, showed "chronic hepatitis with minimal interface activity and mild portal fibrosis (stage 1 of 4)." Ex. 14 at 41; Ex. 15 at 83. But a hepatology follow-up in March 2022 revealed no signs of liver disease, and the latest records filed in this case show no signs of liver disease through November 2023. Ex. 14 at 36–41; Ex. 46 at 6–7.

II. Hearing Witnesses

A. Petitioner's Expert – Dr. Robert Gish, M.D.

Dr. Gish prepared two reports in this case. Gish First Report, dated July 11, 2022, filed as Ex. 16 (ECF No. 18-1) ("First Gish Rep."); Gish Supplemental Report, dated Mar. 21, 2023, filed as Ex. 38 (ECF No. 28-1) ("Second Gish Rep."). He also testified at the hearing. Tr. at 5–116. Dr. Gish was the sole expert presented at trial in support of Petitioner's claim.

Dr. Gish received his M.D. from the University of Kansas, and completed his internship and residency at the University of California, San Diego. Curriculum Vitae, filed on July 11, 2022, as Ex. 37 (ECF No. 20-1) ("Gish CV") at 3. He then completed a fellowship in gastroenterology and hepatology, with a special rotation in liver transplantation, at UCLA. *Id.* He is board-certified in internal medicine and gastroenterology, and has a separate board certification for hepatology that is part of the Certificate of Advanced Qualification in liver transplantation. *Id*. at 2. He is a member of multiple professional societies including the National Viral Hepatitis Round Table, the American Association for the Study of Liver Disease, and the American Liver Foundation. *Id*. at 4. He is a licensed physician in California, Arizona (inactive), and Nevada. *Id*. at 2. He has been active as a clinician and researcher for thirty-six years and has served on the editorial boards of many prestigious journals in his field, including *Hepatology* and the *Journal of Viral Hepatitis.* Gish First Rep. at 1.

Presently, Dr. Gish is a Clinical Adjunct Professor of Medicine at the University of Nevada School of Medicine in both Reno and Las Vegas, and UCSD Skaggs School of Pharmacy and Pharmaceutical Sciences. Gish CV at 1. He is also the Medical Director of the Hepatitis B Foundation, which is the nation's leading nonprofit research and advocacy organization for hepatitis B (HBV). *Id.* Dr. Gish acknowledged, however, that he is not an expert in immunology

(and significantly for purposes of this decision, no such expert was presented by Petitioner). Tr. at 90.

Dr. Gish began his testimony by discussing Petitioner's medical history prior to vaccination—which he deemed not to suggest the existence of developing liver disease or alternative causes. Tr. at 15. For example, she had no physical exam results indicating liver issues, and four normal liver panel tests before receiving the vaccine. *Id*. When a patient has normal liver enzyme tests, "the chance of that person having active liver disease that is hidden in some way is extremely small." *Id*. at 18. He also noted that liver disease patients typically present with symptoms like fatigue, liver pain, jaundice, rashes, and mental confusion. *Id*. at 19. When reviewing Petitioner's medical history, Dr. Gish looked for common causes of liver disease, such as alcoholism, needle sharing, high-risk sexual behavior, and having medical procedures in developing countries—but no such factors were evident. *Id*. at 19–20. Dr. Gish further pointed out that Petitioner had tested negative for Epstein-Barr virus and Hepatitis B and C, which are also significant risk factors. *Id*. at 22.

Dr. Gish admitted that Petitioner's receipt of anti-malarial medication before traveling to Africa was a risk factor for AIH, "but that's typically brief, transient, and doesn't result in autoimmune disease long-term." Tr. at 22. He compared Petitioner's course to that of the patient in a case report whose hepatitis presented acutely after taking an anti-malarial medication. *Id*.; B. Beretta-Piccoli et al*., Atovaquone/Proguanil-Induced Autoimmune-Like Hepatitis,* 1 Hepatology Comm. 293 (2017), filed as Ex. A Tab 10 (ECF No. 25-10) ("Beretta-Piccoli"). The patient evaluated in Beretta-Piccoli displayed symptoms like jaundice and dark urine—obvious clinical signs of liver disease, but unlike Petitioner (at least when her elevated LFT levels were first observed). Tr. at 82; Beretta-Piccoli at 293. Further, Petitioner had already stopped taking all of her medications and supplements after receiving her October 2018 lab results. Tr. at 80. Thus, had the two herbal supplements Petitioner had been taking caused her elevated liver enzymes, the levels should have normalized once she stopped taking them—but she continued to have elevated liver enzymes. *Id*. The above, plus Dr. Gish's view that her disease onset had begun within a few weeks of vaccination, permitted him to conclude that she fit the "ideal profile" for an adverse reaction to the vaccine. *Id.* at 22.

In proposing how the vaccines Petitioner received could have caused AIH, Dr. Gish focused on the measles component of the MMR vaccine as capable of triggering an immune-mediated disease process. Tr. at 38. AIH, he maintained, would usually be initiated by some kind of environmental trigger. *Id.* at 23. The MMR vaccine Petitioner received is an attenuated but live vaccine, which contains components that can "live in the body as a virus" and thereby "manipulate the immune system." *Id.* at 35; First Gish Rep. at 11. As a result, the measles component could temporarily suppress the immune system so that the virus can replicate and persist. Tr. at 36. In an individual with a functioning immune system, regulatory safeguards can turn back on, regulating

the virus and shutting down the immune response. *Id.* But Petitioner's immune response remained chronically activated. *Id.* at 49.

In support, Dr. Gish pointed to filed literature that he maintained established the capacity of the MMR vaccine to suppress the immune response, in ways comparable to the wild measles virus. Tr. at 38–42; R. Nanan et al., *Measles Virus Infection Causes Transient Depletion of Activated T. Cells from Peripheral Circulation*, 12 J. of Clin. Virology 201 (1999), filed as Ex. 31 (ECF No. 19-7) ("Nanan"); T. Munyer et al., *Depressed Lymphocyte Function after Measles-Mumps-Rubella Vaccination,* 132 J. of Infectious Diseases 75 (1975), filed as Ex. 32 (ECF No. 19-8) ("Munyer"); First Gish Rep. at 22 ("[s]ufficient clinical studies confirm that MMR vaccine, even though attenuated, can induce a temporary immune suppression that can last for months").

Here, the suppression of bystander immune cells by the measles virus component of the vaccine allowed an autoimmune cross-reaction to occur. Tr. at 58. Dr. Gish proposed that the pathogenic mechanism responsible for the onset of Petitioner's AIH involved the breaking of self-tolerance to hepatic autoantigens (located on the surface or in the mitochondria of liver cells). *Id.* at 54–55. Normally, a healthy person's immune cells will not cross-react with these hepatic autoantigens, in part from T cell regulation. *Id.* But when T cells are dysregulated, they can incorrectly recognize the hepatic autoantigens on a person's liver cells as being foreign—and attack them. *Id.* at 58. Dr. Gish thus theorized that, in clearing the vaccine-induced measles infection from Petitioner's body, non-measles specific immune cells remained chronically activated thereafter, resulting in a persistent autoimmune condition affecting her liver. *Id*. at 46–49.

In addition, Dr. Gish maintained that the measles component of the MMR vaccine could directly infect immune cells. Tr. at 38; L. Rennick, *Live-Attenuated Measles Virus Vaccine Targets Dendritic Cells and Macrophages in Muscle of Nonhuman Primates*, 89 J. of Virology 2192-2000 (2015), filed as Ex. 33 (ECF No. 19-9) ("Rennick"). This was additional confirmation of the possibility that the measles virus could not only persist in the body post-vaccination but could negatively impact immune function (or at least engage in ongoing immune suppression). First Gish Rep. at 20, 23.

An autoimmune reaction to the measles vaccine was likely compounded by Petitioner's simultaneous receipt of the Tdap vaccine. Tr. at 62.[5] Petitioner's age and records confirmed for

[5] Dr. Gish's initial report also suggested that an adjuvant (a compound included in a vaccine to boost immunogenicity) in the Tdap or MMR vaccine may have also played a role in causing injury. First Gish Rep. at 10, 21, 23. However, at trial he explicitly stepped away from reliance on this possible mechanism. Tr. at 60, 166–68. This was wise—the Program has consistently rejected causation theories relying on the pathogenic impact of vaccine adjuvants. *McKown v. Sec'y of Health & Hum. Servs*., No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (noting that the ASIA theory, "which posits the aluminum vaccine adjuvant as contributing to the purported pathologic immune response, is especially suspect from a scientific standpoint") (citations omitted).

Dr. Gish the fact that Petitioner had previously received a whole cell pertussis-containing vaccine (since replaced by the acellular form due to concerns about adverse events). First Gish Rep. at 12, 21–22. Studies have established the need for pertussis boosters to maintain immunity—and also that production of a specific kind of T helper cell[6] "believed to play an important role in the development of a variety of autoimmune diseases, including autoimmune liver disease" is encouraged in individuals who had previously received the whole cell pertussis form of the vaccine, but then receive as a booster the acellular form. *Id.* at 22; F. Lafdil et al., *Th17 Cells and Their Associated Cytokines in Liver Diseases*, 7 Cellular & Molecular Immuno. 250–54 (2010) (ECF No. 20-2) ("Lafdil").[7] The antigen-specific response generated by this booster vaccine may have amplified the existing response to the measles vaccine, and also itself peaked more quickly (since Petitioner was receiving a booster). Tr. at 62; First Gish Rep. at 23.

As additional support for causation, Dr. Gish relied on a number of case reports (discussed below). He deemed them reasonable evidence of causation under the circumstances, since vaccine injury was rare as a general matter, and given the absence of relevant larger-scale epidemiologic studies. Second Gish Rep. at 1–2. Case reports, he proposed, were at least "signals of issues to be aware of" for clinicians, and they stood as real-world evidence of how a proposed theory might actually unfold. *Id.* at 2, 3 ("if something did happen in another person, then logically the event can happen in this patient"). At the same time, Dr. Gish accepted the possibility of mere coincidence between receipt of a vaccine and development of AIH. *Id.* at 1.

Dr. Gish then briefly discussed an item of literature that he proposed established a secondary explanation for how an individual like Petitioner might experience AIH. *See* S. Subramanian et al., *Postinfectious Autoimmune Hepatitis-Induced Liver Failure: A Consequence of Hepatitis A Virus Infection,* 7 ACG Case Reports J. 1 (2020), filed as Ex. 25 (ECF No. 19-1) ("Subramanian"). But he did not reference Subramanian for its primary findings (which focused on how a hepatitis A wild virus infection could secondarily result in autoimmune hepatitis), but instead for a separate item of literature it discussed, "Vento" (which Petitioner never filed in this case).[8] In Vento, researchers followed family groups that developed autoimmune hepatitis after hepatitis A infections. Tr. at 66–68. The results of the Vento study supported the concept that a genetic disposition could render patients susceptible to AIH, and Dr. Gish felt that this in turn

[6] T helper cells are a kind of immune cell that assist B cells in the production of antibodies. *Zacharski v. Sec'y of Health & Hum. Servs.*, No. 21-317V, 2025 WL 1235431, at *18 (Fed. Cl. Spec. Mstr. Mar. 26, 2025). They serve a different purpose from the type of T cells responsible for directly attacking foreign pathogens.

[7] Although Lafdil is identified on Petitioner's Exhibit List (ECF No. 45-4) as Exhibit 35, it is erroneously marked as Ex. 36 in the filed copy.

[8] *See* Exhibit List, filed December 26, 2023 (ECF No. 45-4). Despite the fact that Vento was never filed as evidence in this case, Dr. Gish characterized it as a "very, very interesting and compelling report," as well as "a[n] excellent paper that would describe what happened to Ms. Exum." Tr. at 67, 69. It is exceedingly difficult to understand why Petitioner (despite demonstrated opportunity) never chose to file this article, if it is in fact so compelling and persuasive.

suggested that Petitioner was likely genetically susceptible as well. *Id*. at 69. But he admitted that no genetic testing had been performed that would corroborate the contention about Petitioner's susceptibility. *Id*. at 96. (And of course, as already noted, the vaccines Petitioner received did not include hepatitis A).

Dr. Gish also reviewed Petitioner's clinical course, deeming it consistent with his causal theory. Tr. at 70. In particular, he opined that Petitioner's AIH symptoms onset began within an acceptable timeframe for an environmental trigger (in this case the vaccine). *Id*. He pointed to Petitioner's fluctuating but elevated liver enzymes (as evidenced by test results obtained between October 2018 and January 2019) as establishing the existence of ongoing AIH, confirmed by the biopsy taken in January 2019. *Id*. at 70–74, 75. Thus, he concluded that "the timing of symptoms, the timing of laboratory tests, the liver biopsy, all fits a classic triggering event and onset of autoimmune disease." *Id*. at 82.

Dr. Gish did not provide a precise proposal for Petitioner's most likely true onset, however. On the one hand, in his first expert report he seemed to embrace the discovery of elevated LFT results, in connection with Petitioner's October 26, 2018 physical exam (occurring 68 days post-vaccination) as a significant evidentiary point establishing the presence of AIH at that time. First Gish Rep. at 24; Second Gish Rep. at 10. But during the hearing, he refined his view, maintaining that onset likely occurred sometime *before* these testing results were obtained. Tr. at 114. Ultimately, Dr. Gish seemed to embrace *any* onset as occurring within ten weeks of vaccination as medically acceptable. *Id.* at 70. But he also seemed to view a timeframe of up to five or six months as acceptable, based on science suggesting that the immune response could remain robust for such a period of time. Second Gish Rep. at 10; Claire-Anne Siegrist, *Vaccine Immunology*, *in* Plotkin's Vaccines 16-34 (S. Plotkin et al. eds., 7th ed. 2018) ("Siegrist"), filed as Ex. 20 (ECF No. 18-5).

On cross-examination, Dr. Gish acknowledged that he has seen patients who developed AIH after traveling, and after taking herbal supplements as well. Tr. at 91–92. He denied that Petitioner's previous small bowel overgrowth ("SIBO") could have caused her AIH, explaining that SIBO is linked to a specific type of AIH which Petitioner did not have. *Id*. at 94. He acknowledged that Petitioner's AIH could have been idiopathic, however, and admitted that an environmental trigger can typically only be identified in half of cases. *Id*. at 95.

When asked about the molecular mimicry aspect of his causal theory, Dr. Gish agreed that he had not identified a specific homology between any amino acid sequences found in proteins of the relevant vaccine components and liver proteins. Tr. at 108. He also attempted to further explain the immune suppression mentioned in his earlier testimony and report, and how the MMR vaccine "both suppresses and activates the immune system" at the same time. *Id*. at 112–16. He specified that the innate immune response is suppressed, leading the adaptive immune response to

compensate. *Id*. at 113. Concurrently, a variety of antigen-presenting cells are activated, producing "off-target effects." *Id*. Then, in a genetically susceptible individual, "[y]ou end up stimulating an arm of the immune system that isn't getting turned off. These are the T-regs that are suppressed in some ways or can[no]t be activated and the immune system goes down this long pathway." *Id*.

B. Respondent's Experts

1. *Jeffrey Crippin, M.D.* — Dr. Crippin authored one report in this case, and testified at the hearing. Crippin Report, dated Oct. 31, 2022, filed as Ex. A (ECF No. 23-1) ("Crippin Rep."); Tr. 117–41.

Dr. Crippin received his medical degree from the University of Kansas, and completed an internal medicine residency at Kansas University Medical Center, where he served as chief resident. Curriculum Vitae, dated Mar. 4, 2024, filed as Ex. E (ECF No. 56-1) ("Crippin CV") at 1–2. He then completed a three-year fellowship in Gastroenterology and Hepatology at the Mayo Clinic in Rochester, Minnesota. Crippin CV at 2. He currently works at the Barnes-Jewish Hospital in St. Louis, and is a Professor of Medicine at the Washington University School of Medicine. *Id*. at 1. He is board certified in internal medicine and gastroenterology, and has received the Certificate of Added Qualification in transplant hepatology. *Id*. at 6–7. He has extensive experience in treating patients with autoimmune hepatitis—he has treated 300–400 patients with the disease over the course of his career. Crippin Rep. at 1.

Dr. Crippin agreed with Petitioner's AIH diagnosis, but denied that the vaccines she received were more likely than not the cause of her illness. Tr. at 123. Rather, he pointed out numerous other potential causal factors in her record, although he was unable to specify one as most likely. *Id*. at 125–27. These factors included Petitioner's international travel in the months prior to her illness; her use of anti-malarial medication and herbal supplements; and a prior history of kidney stones, an IUD, and her SIBO. *Id.* at 125–27, 128–29. Further, she had tested positive for Epstein-Barr virus antibodies in December 2018. *Id*. at 127; Ex. 3 at 207. Dr. Crippin admitted, however, that it was impossible to determine *when* she had Epstein-Barr from that test ("[i]t could have been earlier that year, it could have been five years ago"). Tr. at 127. Although Petitioner experienced fatigue (a symptom of the virus) upon returning from travel, she was not tested for the virus at the time. *Id*. at 128. He also noted that a large number of AIH cases are idiopathic, meaning that no specific trigger can be identified. *Id*. at 130.

Dr. Crippin then discussed one of the case reports Petitioner filed most relevant to this case, pointing out differences from Petitioner's clinical course. Tr. at 131; W. Saliba & M. Elias, *Acute Hepatitis Following MMR Vaccination*, 16 Euro. J. of Internal Med. 379 (2005), filed as Ex. 21 (ECF No. 18-6) ("Saliba"). Although Saliba involved the MMR vaccine, it featured a patient who experienced *acute* hepatitis rather than AIH, with a post-vaccination onset of two weeks (not the

six to eight weeks likely in this case). Tr. at 131–32; Saliba at 379. The Saliba patient had also recently given birth, putting her at greater risk of viruses due to pregnancy-related immune suppression, and no liver biopsy was performed to help determine the cause of her hepatitis. Tr. at 131–32; Saliba at 379.

Dr. Crippin also noted that he had been unable to locate any controlled studies showing a link between AIH and either the MMR or Tdap vaccines. Tr. at 130. And he similarly was unaware of studies establishing that the combined administration of both vaccines at once constituted a risk factor for AIH. *Id.* at 133. On cross, he reiterated his prior testimony that he could not determine when Petitioner was infected with Epstein-Barr virus, and that there were no other infections noted in her records. *Id*. at 135. Ultimately, Dr. Crippin declined to identify which of the various factors he deemed the most likely cause, and stated again that this could be an idiopathic case. *Id.* at 137–38, 140–41.

2. *Andrew MacGinnitie, M.D., Ph.D.* — Dr. MacGinnitie wrote one report in this case, and testified at the hearing. MacGinnitie Report, dated Oct. 27, 2022, filed as Ex. C (ECF No. 23-3) ("MacGinnitie Rep."); Tr. at 141–92.

Dr. MacGinnitie is the Chief of the Division of Allergy, Asthma, and Immunology at Children's Hospital of Wisconsin, and a Professor of Pediatrics at Medical College of Wisconsin. Tr. at 142–43. He graduated from the University of Chicago Pritzker School of Medicine with both an M.D. and a Ph.D. from the Department of Pathology. Curriculum Vitae, dated Oct. 31, 2022, filed as Ex. D (ECF No. 23-4) ("MacGinnitie CV"). He then completed a residency in pediatrics in the Boston Combined Residency Program, training at Boston Children's Hospital and Boston Medical Center, followed by an allergy/immunology fellowship at Boston Children's Hospital. *Id.* at 1. He is board certified in both allergy/immunology and pediatrics. *Id.* at 11. He maintains an active clinical practice seeing more than 1600 patients annually and has extensive experience in caring for children and adults with a variety of immunologic diseases, including reactions to vaccines. MacGinnitie Rep. at 2. Dr. MacGinnitie also performs research and has published articles in a number of areas related to allergy/immunology including food allergy, vaccine reactions, and primary immunodeficiency. *Id.* Dr. MacGinnitie was the sole immunology expert to offer testimony in this case.

Dr. MacGinnitie opined that the vaccines Petitioner received had no likely relationship to her AIH. Tr. at 148. First, he criticized Dr. Gish's reliance on case reports (in the absence of epidemiological studies connecting AIH and the Tdap and MMR vaccines). *Id.* at 150. In his view, case reports cannot reliably connect a vaccine to an illness because they do not provide an accurate comparison with the baseline rate of an illness in the general population. *Id.* Further, none of the case studies filed involved instances of simultaneous administration of the Tdap and MMR vaccines. *Id.* at 151; *see*, *e.g.*, M. van Gemeren et al., *Vaccine-Related Autoimmune Hepatitis: The*

Same Disease as Idiopathic Autoimmune Hepatitis? Two Clinical Reports and Review, 52 Scandinavian J. of Gastroenterology 18 (2017) (involving a combination of Tdap and hepatitis A vaccines), filed as Ex. 26 (ECF No. 19-2) ("van Gemeren"); *see also* P. Perumalswami et al., *Vaccination as a Triggering Event for Autoimmune Hepatitis*, 29 Seminars in Liver Disease 331 (2009) (discussing hepatitis A and yellow fever vaccines), filed as Ex. 27 (ECF No. 61) ("Perumalswami"). And although Saliba did involve the MMR vaccine, the relevant patient only experienced an acute form of hepatitis. Tr. at 152; Saliba at 379. In another case report offered by Dr. Gish, the patient received six vaccines at the same time, and although the MMR vaccine was one of them, it was impossible to isolate the effects of only one or two of the listed vaccines. Tr. at 152–53; G. Veerappan et al., *Vaccination-Induced Autoimmune Hepatitis,* 50 Digestive Diseases and Sci. 212 (2005), filed as Ex. 24 (ECF No. 58-1) ("Veerappan").

Second, Dr. MacGinnitie took issue with Petitioner's invocation of molecular mimicry as a possible mechanism for vaccine-induced AIH. Tr. at 156. After explaining the concept briefly, he noted that Dr. Gish had not identified any specific homology (meaning molecular similarity) between amino acid sequences in the vaccines' protein components and the liver cell antigens where an autoimmune cross-reaction would begin or occur—a crucial starting point if molecular mimicry was to stand as a reasonable explanation for Petitioner's injury. *Id*. at 158. Moreover, even a demonstration of homology would not be sufficient to prove the theory in Dr. MacGinnitie's view, as there is a "massive overlap between microbial and human proteins" in nature that does not commonly result in autoimmunity. *Id*. He also criticized Petitioner's reliance on bystander activation of secondary/nonspecific immune cells as a possible disease mechanism, noting that articles filed in the case specific to AIH did not consider this to be a pathologic explanation. *Id.* at 160; C. Mack et al., *Diagnosis and Management of Autoimmune Hepatitis in Adults and Children: 2019 Practice Guidance and Guidelines from the American Association for the Study of Liver Diseases*, 72 Hepatology 671 (2020), filed as Ex. A, Tab 1 (ECF No. 25-1) ("Mack"). Beyond this, Petitioner had not displayed clinical signs of significant inflammation, which would have occurred shortly after vaccination had such an autoimmune response been underway. Tr. at 159.

Dr. MacGinnitie also addressed Petitioner's argument that her immune system was likely suppressed by the measles vaccine, allowing an autoimmune response to occur. He noted the absence of strong evidence showing that the measles *vaccine* (as opposed to the wild virus) suppresses the immune system pathologically. Tr. at 162; MacGinnitie Rep. at 8–9. He deemed articles filed to support this contention as lacking in clinical value and/or outdated. Tr. at 162–64; Nanan (published in 1999); Munyer (published in 1975). Up-to-date clinical manuals relied upon by treaters, however, acknowledge the immune-suppressive nature of the *wild measles virus*, but not the vaccine (the receipt of which functions to *prevent* this immune suppression in the first place). Tr. at 165–66; American Academy of Pediatrics, Red Book (2021): Report of the Committee on Infectious Diseases 503 (32d ed. 2021), filed as Ex. C, Tab 4 (ECF No. 26-4) ("AC of Pediatrics"); *see also* M. Mina, *Measles, Immune Suppression, and Vaccination: Direct and*

Indirect Nonspecific Vaccine Benefits, 74 J. Infection S10, S15 (2017), filed as Ex. C, Tab 3 (ECF No. 26-3) ("Mina") (noting the benefit of measles vaccine in blunting the immunosuppressive character of a wild measles virus infection). Dr. MacGinnitie also noted that a theory dependent on immunosuppression did not fit with AIH's likely pathogenesis. Since AIH is considered autoimmune in character, it reflects an aberrantly *overstimulated*/overactive immune process—not one that has been suppressed (allowing *another* opportunistic infectious process might occur). Tr. at 162.

Dr. MacGinnitie challenged Dr. Gish's argument that whole cell pertussis[9] could itself stimulate a class of T-helper cells (which encourage the production of certain proinflammatory cytokines)—and thus, because Petitioner had likely received the whole cell pertussis vaccine as a child, the Tdap booster she had received in 2018 might have increased the possibility of a comparable immune memory response. Tr. at 169. He acknowledged the importance of the relevant T-helper cells in fighting bacterial and fungal infections, but deemed the re-stimulation of them unlikely to cause autoimmune disease. *Id.* at 170. Thus, although Petitioner had offered a study showing that patients vaccinated with whole cell pertussis produced more inflammatory cytokines than those initially vaccinated with acellular pertussis, the study did not also establish that the produced cytokines rose to levels anywhere near sufficient to propagate an autoimmune inflammatory environment. *Id*. at 171–72; R. da Silva Antunes et al., *Th1/Th17 Polarization Persists Following Whole-Cell Pertussis Vaccination Despite Repeated Acellular Boosters*, 128 J. Clin. Investigation 3853 (2018), filed as Ex. 34 (20-1) ("da Silva Antunes"). And since Petitioner had not been tested for these cytokines, it was pure speculation to propose she likely possessed them in pathologic levels after vaccination. Tr. at 173.

Dr. MacGinnitie concluded with a brief consideration of the onset interval of one to five months Dr. Gish proposed for vaccine-induced AIH. Tr. at 174. Although he opined that case reports should carry little evidentiary weight, he noted that the case reports Dr. Gish cited showed documented hepatitis (not simply the first symptoms) beginning within a far shorter timeframe: ten to thirty days of vaccination. *Id*.; *see*, *e.g.*, T. Sasaki et al., *Autoimmune Hepatitis Following Influenza Virus Vaccination: Two Case Reports*, 97 Med. 1 (2018), filed as Ex. 28 (ECF No. 19-4) ("Sasaki") (documenting one week onset and one month onset). Other items of literature Petitioner referenced for a shorter onset timeframe involved distinguishable diseases or vaccines. Tr. at 175–76; L. Schonberger et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 Am. J. of Epidemiology 105, filed as Ex. 39 (ECF No. 28-2) ("Schonberger") (discussing the flu vaccine and Guillain-Barré syndrome ("GBS")).

[9] The now largely-discontinued DPT vaccine included whole cell pertussis, but the version administered today (Tdap) employs an acellular form of pertussis thought to be less likely to cause certain side effects. *See Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1375 n.1 (Fed. Cir. 2009) for discussion of case law on the safety concerns prompting the switch to an acellular formulation.

III. Review of Filed Literature

Ample Program authority establishes that special masters are not required to describe and/or analyze in a written decision every item of literature filed in support of or against a claim. *See*, *e.g.*, *Snyder v. Sec'y of Health and Hum. Servs.*, 36 Fed. Cl. 461, 466 (1996), *aff'd*, 117 F.3d 545 (Fed. Cir. 1997). Indeed, the failure to specifically mention a filed item in a decision does not allow for the inference that it was not included in the special master's overall weighing of evidence. *Hazlehurst v. Sec'y of Health and Hum. Servs.*, 604 F.3d 1343, 1352 (Fed. Cir. 2010) (noting that a reviewing court presumes that the fact finder has considered all of the material in the record, regardless of whether it is individually mentioned in his or her decision); *see also Moriarty v. Sec'y of Health & Hum. Servs.,* No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

There is also the fact that this matter went to a live hearing. A hearing provides the parties the opportunity to succinctly but fully explain their take on a case in the best possible manner. In holding hearings, special masters reasonably focus their attention on the items of literature the parties deem most worthy of verbal mention during an expert's testimony. Presenting an entitlement claim is not a game of "hiding the ball," where a claimant seeds the record with numerous items of literature, references only some at hearing, and then later objects on appeal when certain filed items do not receive a full airing in a written decision. *Echols v. Sec'y of Health & Hum. Servs.,* 165 Fed. Cl. 9, 12 (2023) ("the evidence heard by the special master should be the "main event" rather than a mere "tryout").

The Remand Order nevertheless has identified some items of literature as requiring more discussion, in order to disclose the full extent of my reasoning. Remand Order at 40–44. Accordingly—and because I ultimately reach the same conclusion that I initially did—I will provide a detailed summary of every single item of literature filed by Petitioner in this case.[10]

[10] Petitioner filed a relatively modest 22 articles or studies. *See* Petitioner's Exhibit List as of December 26, 2023, filed Feb. 9, 2024 (ECF No. 53-2).

A. *Petitioner's Literature*[11]

1. Mack

Both sides offered this article. Mack provides an overview of AIH, and guidelines for its diagnosis and treatment. *See generally* Mack. Petitioner offered Mack to explain AIH's immunopathogenesis, and to establish that Petitioner was properly diagnosed with AIH. First Gish Rep. at 7. But Petitioner's diagnosis is not questioned in this case. To the extent Mack bears on causation, it is discussed in greater detail in the analysis section of this Decision.

2. U. Christen & E. Hintermann, *Pathogens and Autoimmune Hepatitis*, 195 Clin. and Experimental Immuno. 35-51 (2019), filed as Ex. 18 (ECF No. 18-3) ("Christen").

Petitioner has offered Christen in support of her argument that multiple viruses, including the measles virus, are implicated in the pathogenesis of AIH. First Gish Rep. at 10. Christen is a review article discussing some of the possible environmental triggers for AIH. Christen at 35. Unquestionably, the article mentions "pathogen infections and vaccinations" as possible environmental factors that can interact with a preexisting genetic risk factor. But Christen solely discusses wild viral or bacterial pathogens, focusing on the most obvious (hepatitis viruses) or the Epstein-Barr virus. *Id.* at 40–43.

In a brief section addressing "other pathogens" possibly capable of triggering AIH, Christen notes the existence of "two studies from the 1980s" in which AIH patients were found to possess evidence of a measles virus infection, but adds that the studies relied on diagnostic definitions for AIH that would no longer be valid—and that another epidemiologic study had subsequently revealed "no significant difference" between a studied population and AIH patients who generated measles virus antibodies. Christen at 43.[12] At most, Christen posits that some case reports involving AIH after a measles infection allow for the hypothetical possibility of measles virus as an infectious trigger—although the sole reference for this case report is the same article mentioned by Dr. Gish but never filed, Vento. *Id.* at 43 n.111. Christen also acknowledges that little evidence supports the hypothesis that AIH is driven by molecular mimicry, although (unlike Mack) Christen does mention bystander activation, at least as a theoretical mechanism. *Id.* at 37, 38.

[11] I refer to an article's abbreviated title in all cases where the item was previously defined in this manner in this Decision.

[12] Petitioner filed one of the two 1980s articles referenced herein—Robertson (*see* Christen at 43 n.109), plus the epidemiologic article that Christen notes undermined a measles virus—AIH connection (Mieli-Vergani I—Christen at 43 n.110). Both are discussed below.

3. C. Benn et al., *A Small Jab – A Big Effect: Nonspecific Immunomodulation by Vaccines*, 34 Trends in Immuno. 431-39 (2013), filed as Ex. 19 (ECF No. 18-4) ("Benn").

Petitioner cites Benn to support her contention that vaccines can activate nonspecific bystander immune cells. First Gish Rep at 10. Benn notes that "[r]ecent epidemiological studies have shown that, in addition to disease-specific effects, vaccines against infectious diseases have nonspecific effects on the ability of the immune system to handle other pathogens." Benn at 431. The authors of Benn explain that each exposure to infection or vaccination leaves an imprint on a person's immune system, which can affect future innate and adaptive responses to new pathogens. *Id.* at 433. This concept of "heterologous immunity" could explain why vaccines may have nonspecific effects—because the vaccines encode antigens that cross-react with other pathogens. *Id.* The authors conclude, therefore, that cross-reactive T-cell-mediated heterologous immunity is likely a common determinant in the pathogenesis of infections. *Id.* at 433–34.

Benn, however, primarily focuses on the *positive* rather than pathologic indirect effects of vaccination. In Benn's discussion of the measles vaccine, for example, the authors emphasize the vaccine's observed survival benefits. Benn at 432, 436 ("[e]xisting studies suggest a general pattern, namely that the live vaccines: [including measles vaccine] are associated with *beneficial* nonspecific effects, leading to reduced all-cause mortality . . . ." (emphasis added)). Measles-vaccinated children have substantially lower mortality than can be explained by measles-related deaths, suggesting that the vaccine comes with a host of nonspecific beneficial effects. *Id.* at 432. Benn does not purport to state *how* vaccines most likely induce these nonspecific effects, but it *does not support* the conclusion that the live vaccines it discusses have pathologic potential due to their nonspecific effects (at best allowing for the need to identify when those effects *could* be detrimental, so that concurrent therapeutic interventions could be developed to inhibit negative outcomes). *Id.* at 437.[13] And Benn says nothing specific about AIH and its purported vaccine association.

4. Siegrist

Siegrist is an excerpt from a larger publication, and it provides a general explanation for how a vaccine activates the immune system. Among other things, it highlights the role of T cells in the induction of high-affinity antibodies and immune memory. Siegrist at 16. It also notes that live vaccines (like MMR) "rapidly disseminate throughout the vascular network to reach their target tissues" in a pattern that is "very similar to that occurring after a natural infection." *Id.* at 20.

[13] At most, Benn contains a discussion of some instances where "detrimental heterologous immunity can lead to severe immunopathology." Benn at 434. But the instances discussed involve studies featuring immunization of animal subjects with different vaccines, resulting in other forms of disease not bearing on this case, or where receipt of one form of a vaccine can "lock" an immune response to subsequent forms, causing a vaccine to have "detrimental effects on the outcome of secondary infections." *Id.* at 435.

But Siegrist also, in Petitioner's estimation, has something to say about the impact of receipt of the non-live, Tdap vaccine. She received Adacel, a Tdap booster, and that vaccine was expected to activate Petitioner's immune memory cells and result in a rapid increase of antibody titer. First Gish Rep. at 23. According to Figure 2.3 in Siegrist, booster exposure to antigen reactivates immune memory and results in an increase of IgG antibody titer within seven days of receipt. Siegrist at 24. Short-lived plasma cells responsible for production of antibodies thereafter maintain peak antibody levels for a few weeks. *Id.* And six months after booster vaccination, long-lived plasma cells reach survival niches in the bone marrow and continue to produce antigen specific antibodies. *Id.*

Petitioner cited to Siegrist to support her arguments about the medically-acceptable timeframe in which her AIH manifested post-vaccination. First Gish Rep. at 24. Petitioner started to experience fatigue in the weeks after vaccination, but then continued to suffer increased symptoms in the five months up to her formal diagnosis in January 2019. Petitioner argues that this symptom progression fits within the one to five-month timeframe suggested in Siegrist. First Gish Rep. at 24.

5. Saliba

As discussed above, Petitioner has offered Saliba to support her argument that the MMR vaccine can cause a person to develop hepatitis. Saliba is a one-page "letter to the editor" which reports an instance in which a 24-year-old woman developed acute hepatitis two weeks after receiving the MMR vaccine, with her hepatitis thereafter resolving within a month (as reflected by LFT results). Saliba at 379. The sequence of events, together with the lack of potential causes for elevated liver enzymes, "strongly suggest[ed]" to its authors that the patient's development of hepatitis was related to vaccination. *Id.* The proposed mechanism was a cytotoxic, cell-mediated, immunological response with injury of the hepatocytes. *Id.* But Saliba notes (at least as of the time of its publication) that "there is still no evidence that [the vaccine's viral components] are directly cytopathic to the hepatocytes," although it referenced an older article (not filed in this case) purportedly establishing that biopsies of patients with rubella infection have revealed "necrosis of liver cells" and suggesting that "cytotoxin T cell lymphocytes play an important role in liver injury." *Id.* Saliba's short onset timeframe is self-evidently distinguishable from this case.

6. J. McMahon et al., *Measles Vaccine Virus RNA in Children More Than 100 Days after Vaccination*, 11 Viruses 636-48 (2019), filed as Ex. 22 (ECF No. 18-7) ("McMahon").

McMahon is another instance of an article cited by Petitioner as supporting a point that the article itself only stands for secondarily. Noting the general dangers of a wild measles infection (especially because of its immunosuppressive character), and the corresponding benefit of the measles vaccine, McMahon's authors evaluated some instances of "extremely delayed" detection

of the measles vaccine in a sample of more than 9,000 children. McMahon at 638-39. Ordinarily, RNA from a measles virus strain found in the vaccine is commonly detected for up to *at least* 14 days post-vaccination. *Id.* at 638, 643. But McMahon confirmed that the strain can be detected between 100- and 800-days post-vaccination, albeit based on a subset of only *eleven* samples. *Id.* at 638.

McMahon's findings were deemed consistent with the understood persistence of a wild virus measles infection, and its authors also noted the significance of the fact that the vaccine component RNA was specifically identified in the respiratory tract of the studied patients (underscoring the degree to which lymphoid tissue—to which a natural infection would spread as immune cells encountered the virus—was a likely situs for the virus to remain in the body). McMahon at 643. But its authors in no way implicated the vaccine as dangerous or pathologic in connection with its immune persistence (except in the limited circumstance of a person known to be immunodeficient or receiving immunosuppressive treatments). *Id.* Indeed, McMahon's authors expressly observed that the vaccine remained safe and highly important in the prevention of measles, and that the exceedingly small sample size relied upon for its findings prevented drawing larger conclusions from the study. *Id.* at 644.

Dr. Gish relied in part on McMahon for the contention that persistence of the measles virus in Petitioner could have later contributed to her development of chronic liver inflammation. Tr. at 50; First Gish Rep. at 24. But McMahon certainly does not equate persistence with pathogenicity—active or potential. Indeed, as Dr. MacGinnitie observed, McMahon did not show that the studied children actually had an infection in their liver—the measles vaccine virus RNA was only present in their *respiratory secretions*. Tr. at 176. And although the eleven samples had more often than not presented pre-detection with some infectious-like symptoms, McMahon's authors noted that they could not confirm whether other, concurrent viral infections were not explanatory—and even more importantly, that "detection of [vaccine component measles RNA] alone does not allow for any assessment of whether infectious virus is present." McMahon at 644.

7. P. Berry & G. Smith-Laing, *Hepatitis A Vaccine Associated with Autoimmune Hepatitis*, 13 World J. of Gastroenterology 2238-39 (2007), filed as Ex. 23 (ECF No. 18-8) ("Berry").

Berry is a case report in which a 56-year-old man developed acute liver injury consistent with autoimmune hepatitis ten days after he received the hepatitis A vaccine. Berry at 2238. Notably, the patient in Berry had also been diagnosed with acute hepatitis five months before the administration of the vaccine. *Id.* This case is thus distinguishable from Petitioner's case, involving not only an entirely different vaccine—the hepatitis A vaccine—but a patient who had experienced a *relapse* of hepatitis post-vaccination.

8. Veerappan

Veerappan is another case study offered for the general proposition that autoimmune hepatitis is associated with vaccination. First Gish Rep. at 16. In Veerappan, a 35-year-old male developed severe flu-like symptoms *one week* after receiving multiple vaccinations—typhoid, hepatitis A, oral polio, diphtheria/tetanus, and MMR. Veerappan at 212. A liver biopsy performed one month after initial presentation showed severe chronic hepatitis, which was deemed to be consistent with AIH. *Id.* The authors concluded that AIH may be a complication in patients who receive multiple vaccinations simultaneously. *Id.* at 213. But Veerappan's authors provide no explanation for the significance of the receipt of so many vaccines at once, deeming them "a novel inciting event" worthy of consideration (but not shown to be likely pathogenic). *Id.* And Petitioner's onset was not nearly so quick in this case.

9. Subramanian

Subramanian is yet another, facially-inapposite case report. Here, a 45 year-old woman who had been diagnosed with a hepatitis A infection was hospitalized a month later, and her presenting symptoms and lab work were later deemed consistent with AIH. Subramanian at 1–2. Thus, the studied individual had not received any vaccine at all (let alone the MMR or Tdap vaccines), and was suspected to have developed AIH from her prior *hepatitis* infection (evidence of which was detected after her disease onset and which "remained positive" during her treatment. *Id.* at 2.

Petitioner nevertheless (as noted above) relied on Subramanian, with Dr. Gish maintaining that it stood as evidence of the genetic susceptibility for AIH. Tr. at 69. But as noted above, Dr. Gish's primary interest in Subramanian arises from its reference to an unfiled article (Vento) in which two relatives developed autoimmune hepatitis within five months of an acute hepatitis infection. Subramanian at 2 n.4. The authors concluded that a genetic disposition (specifically, a type of T cell defect) made the patients susceptible to AIH following the impact of infection. *Id.* In Dr. Gish's view, Petitioner similarly likely suffered some kind of "T cell defect" due to genetic susceptibility, like the patients in Vento, but triggered here by a different acute immunologic stimulus. *Id.* at 69. But Dr. Gish later admitted that no genetic testing had been performed that would corroborate Petitioner's susceptibility. *Id*. at 96. And Subramanian itself does not suggest that exposure to a measles wild virus or vaccine component could have the same impact on a genetically susceptible individual as a hepatitis A infection.

10. van Gemeren

van Gemeren discusses two women in their 20s who developed AIH after receiving various vaccines. van Gemeren at 18–19. One developed AIH a month after receiving vaccines against hepatitis A and hepatitis B. *Id.* at 18. The other developed AIH ten days after she was vaccinated

against hepatitis A, diphtheria, whooping cough, and tetanus. *Id.* at 19. The article suggested that vaccination, especially vaccination against hepatitis A, may trigger AIH in genetically susceptible individuals. *Id.* at 21–22. However, van Gemeren acknowledges that no conclusive evidence has been found for a causal association between vaccines and the occurrence of autoimmune diseases. *Id.* at 21. Otherwise, this case report does not involve the MMR vaccine (the focus of Petitioner's causation theory), does not discuss the impact of the tetanus-containing vaccine to any significant degree, and does a better job underscoring the possible impact of hepatitis-oriented vaccines.

11. Perumalswami

The Perumalswami case report discussed a 31-year-old woman who developed autoimmune hepatitis 11 days after receiving hepatitis A and yellow fever vaccines. Perumalswami at 331. After vaccination and prior to developing symptoms, she had traveled to Nigeria for five days. *Id.* Perumalswami's authors raised the possibility that these two vaccines may have been a triggering agent for AIH (but admitted that this was a novel finding). *Id.* at 333. Like the other case reports discussed above, however, this article says less about the potential pathogenicity of the vaccines at issue than it does about the risks of a hepatitis vaccine.

12. Sasaki

Sasaki is the final case report offered by Petitioner to substantiate a vaccine-AIH relationship. In Sasaki, two female patients presented with AIH within one week and one month, respectively, after receiving the flu vaccine. Sasaki at 1–2. The authors admitted that a causal link could not be established, but hypothesized that the flu vaccine could trigger the development of AIH, given the possible link between immunization and other autoimmune diseases. *Id.* at 3. But as Dr. MacGinnitie pointed out, flu vaccine administration is so widespread that it is very likely that the development of AIH after vaccination in these cases was mere coincidence. Tr. at 155. And yet again—Sasaki, like prior case reports, does not involve the relevant vaccines in this case.

13. D. Robertson et al., *Persistent Measles Virus Genome in Autoimmune Chronic Active Hepatitis*, 330 The Lancet 9-11 (1987), filed as Ex. 29 (ECF No. 19-5) ("Robertson").

Robertson, a study from 1987, involves the measles virus and its putative association with "chronic active hepatitis." Robertson at 9. 12 out of 18 studied patients with confirmed autoimmune chronic active hepatitis were found also to possess the measles virus genes. *Id.* at 10. Robertson's authors concluded that the presence of high-titer antibodies to the measles virus in patients with AIH "suggests that these particular viruses may have a causal role in the condition." *Id.* They also surmised that the patients' exposure to measles resulted in a persistent incomplete infection occurring as a result of continued production of measles antigen and ineffective elimination of the virus. *Id.* at 11.

The Robertson study briefly mentions the measles vaccine but does not offer a potential causal mechanism, and suggests even that the measles vaccine might have *prevented* some instances of measles-caused AIH (resulting in onset only in older cohorts). Robertson at 10. In addition (and as noted in my discussion of Christen above), there is reason to doubt whether the defined group of individuals with "autoimmune chronic active hepatitis" would be suffering from what is currently understood to be AIH. Otherwise, Robertson's authors admitted that the finding of persistent measles virus in the small set of subjects could simply be attributable to an "epiphenomenon"[14]—meaning a secondary effect arising from, but not causal of, a primary process (and thus not causal of the studied, hepatitis-like illness).

14. G. Mieli-Vergani et al., *Measles and Autoimmune Chronic Active Hepatitis*, 334 The Lancet 688 (1989), filed as Ex. 30 (ECF No. 19-6) ("Mieli-Vergani I").

Petitioner deemed Mieli-Vergani I to substantiate an association between the measles vaccine and AIH. Mieli-Vergani I's authors (in a single-paragraph summary[15] published in the *Lancet*) mentioned a study observing that 8 out of 12 children with autoimmune hepatitis had received the measles vaccination and had tested positive for low titers of measles antibody. Mieli-Vergani I at 688. The authors surmised that there could be a causal link between measles and autoimmune hepatitis, at least in childhood. *Id;* First Gish Rep. at 19. But the cursory discussion of these findings prevents any greater scrutiny of their reasonableness. And as noted expressly in Christen—published 30 years later, and thus with the benefit of further study (as well as perhaps a full copy of the article)—Mieli-Vergani I did not find the existence of the measles antibody titers significant, when comparing sick individuals with a control, healthy population—thereby reducing the evidentiary value of this somewhat-stale article. Christen at 43.

15. Nanan

Nanan's authors sought to investigate the impact of measles infection-induced immune suppression (which has also been observed to a lesser extent in vaccination) by seeking to analyze T cells extracted from serum of four pediatric subjects with measles infections, plus four older individuals who had received the measles vaccine. Nanan at 202. In particular, it sought to observe the expression of leukocyte function-associated antigen 1 (LFA-1)—a cell surface protein found on human T cells, and deemed important to a functioning immune process. *Id.* at 201–02.

[14] "Epiphenomenon" is defined as "an accessory, exceptional, or accidental occurrence in the course of an attack of any disease." *Epiphenomenon*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=16902&searchterm=epiphenomenon (last visited on May 27, 2025).

[15] Petitioner has not filed a full version of Mieli-Vergani I.

Nanan found that there was a "remarkable loss" of LFA-1-bright cells during natural measles infection, and after measles vaccination. Nanan at 204–05. The findings suggested to the authors that sequestration of T cells is a feature of the measles virus infection and vaccination. *Id.* at 209. This is problematic, since the integration and control of immune responses depend on regulated trafficking of lymphocytes. *Id.* The disruption of lymphocyte trafficking by random distribution of T cells in all areas of lymphoid organs could impair host defenses and result in immune suppression. *Id.*

Petitioner cited Nanan to support her contention that the measles component of the MMR vaccine could initiate immune dysfunction, leading in some form to an autoimmune response (although Nanan itself does not discuss such secondary disease in the context of measles-induced immunosuppression). Tr. at 40–41; First Gish Rep. at 19. While Dr. Gish admitted that there is no evidence in this case that Petitioner likely ever suffered from a prolonged measles vaccine-induced suppression, he contended that this was not necessary to meet Petitioner's burden of proof on causation. Tr. at 45–46. And it was likely in any event that immune activation persisted even if Petitioner's system had cleared measles post-vaccination. *Id.* at 49. In response, Dr. MacGinnitie emphasized that the authors of Nanan did not provide evidence that the observed decrease of LFA-1 was clinically meaningful, nor did they offer evidence that the small group of patients they considered had experienced any actual immune suppression sufficient to cause disease. Tr. at 163.

There is no reason to doubt the validity of Nanan's specific findings, and I deem its methodology reliable. Indeed, its findings are consistent with the already-understood immunosuppressive impact of the wild measles virus. But Nanan cannot be read to support the conclusion that receipt of a measles-containing vaccine is likely *pathologic* for this same reason – or that AIH is more likely in the context of a prior measles vaccination. It thus stands as somewhat weak evidence overall for the contention that the MMR vaccine has the capability to produce disease-encouraging immune suppression.

16. Munyer

Munyer is a 1975 study that sought to consider the impact of receipt of the version of MMR vaccine used at the time on T cell responses in the blood of children. Sera was drawn from an unspecified number of vaccinated children (pre and post-vaccination), then stimulated ex vivo. Munyer at 75–76. An impairment in lymphocyte response to stimulation with antigen was observed in the vaccinated samples (via an in vitro test), with the impairment lasting for one to five weeks post-vaccination. *Id.* at 77. From this, Munyer's authors concluded that the MMR vaccine could cause a depression of lymphocyte function - although the degree of impairment was deemed likely more pronounced for an active infection than vaccination. *Id.* at 77–78.

Munyer also, however, noted the following:

> no significant alteration was observed in the absolute number of lymphocytes in the peripheral blood of vaccinated subjects, either in comparison with base line (before vaccination) counts or with counts from a group of unvaccinated controls. Similarly, no decrease in either the percentage or the absolute number of peripheral blood T-lymphocytes was observed after vaccination.

Munyer at 76. Thus, although experimental stimulation of blood samples revealed an impairment in immune cells, *actual, in vivo vaccination did not*. It is the results of the latter that are ultimately in contention in this case.

Petitioner cites Munyer for the same reason as Nanan—to support her argument that the measles vaccine could have induced immunosuppression in Petitioner, leading to the development of AIH. First Gish Rep. at 19. Dr. MacGinnitie criticized this article during his testimony, however, noting that it was 50 years old (and hence outdated), and claiming that its authors used "rather primitive techniques." Tr. at 163. More importantly, however, Dr. MacGinnitie observed that Munyer did not provide evidence that the immunosuppression observed was meaningful from a clinical standpoint (as opposed simply to an observed, absolute phenomena). *Id.* The above-referenced block quote citation from the article directly corroborates this opinion.

My reaction to Munyer is consistent with my reading of Nanan. While Munyer's findings may narrowly be based on a methodologically-reliable experiment, the article stands only as a single (and facially-outdated) "brick" in the causation theory "wall" Petitioner seeks to build—and it does not itself establish vaccine-related pathology. Rather, it constitutes additional evidence that the measles component of the MMR vaccine may cause an experimentally-observable amount of immune suppression—but whether that is of a magnitude sufficient to result in an autoimmune disease is not addressed. And it cannot be concluded from Munyer that the vaccine *when actually administered in vivo* does cause observable immune suppression, and/or has pathologic outcomes. Munyer's own findings suggest the opposite.

17. Rennick

Rennick noted that despite the widespread use of measles vaccines, "little is known about the attenuation profile of the vaccine virus or what cells it targets upon vaccination." Rennick at 2197. Accordingly, Rennick's authors sought to compare the immunogenicity of the vaccine compared to its biologic viral parent, specifically by looking to identify the vaccine's antigenic targets after administration by injection. *Id.* The study (using animal subjects) determined that infected cells after vaccination are predominantly macrophages and dendritic cells in subcutaneous tissues, found abundantly at the site of injection (rather than muscle cells). *Id.* at 2199.

Petitioner has cited Rennick as establishing that the measles vaccine virus strain likely infected Petitioner's immune cells. Tr. at 38. But the means by which the immune system takes up the vaccine does not also lead to the conclusion that pathology was an expected, or even possible, outcome *due* to vaccination. And Rennick says other things about the impact of a measles infection that are inconsistent with Petitioner's theory. For example, Rennick notes that the measles wild virus has a much shorter, preclinical latency period after infection (10 to 14 days) than what is proposed in this case, undermining Petitioner's general argument that once vaccinated, pathology remains possible months later. Rennick at 2192. Rennick also underscores the principal danger of a wild measles infection—the increase in "susceptibility to opportunistic infections"—in addition to direct risk from the *infection itself* (and here, the evidence that Petitioner's AIH is attributable to an opportunistic hepatitis infection is wholly lacking).

18. da Silva Antunes

da Silva Antunes constitutes one of the few articles offered in this case to link the Tdap vaccine to Petitioner's injury. Its authors sought mainly to better understand the effects of the transition from the use of whole cell pertussis-containing vaccines to the acellular form (which in the U.S. replaced whole cell pertussis, due to concerns that the former version was associated with adverse events). da Silva Antunes at 3856. Accordingly (and in the wake of evidence that pertussis disease was increasing even in places where the acellular form was used), da Silva Antunes's authors attempted to compare the immune responses of children who received whole cell pertussis originally versus those who first received the acellular form, comparing both groups after they received an acellular pertussis booster. *Id.* at 3855. The study revealed that subjects who had been previously vaccinated with whole cell pertussis produced more of a specific form of T helper cell (Th17 cells). *Id.* at 3856–58. In effect, despite the adverse events associated with the largely-discontinued whole cell pertussis, receipt of that form of the vaccine was deemed to impart greater immunity overall. *Id.* at 3869–70.

Petitioner cited da Silva Antunes to show how an increase in Th17 cells could lead to autoimmune hepatitis. Tr. at 169–70. Dr. Gish reasoned that Th17 cells are critical to regulate both protective and pathogenic immune responses. First Gish Rep. at 22. Because Petitioner was likely administered the whole cell pertussis vaccine as a child, she would produce more Th17 cells after she received the Tdap booster vaccine. *Id.* Dr. MacGinnitie argued in response, however, that Dr. Gish had never explained how the production of Th17 cells would actually result in AIH (specifically by causing damage to cells in the liver). Tr. at 173. And Petitioner's Th17 levels were never tested, so there was no way to know if they were elevated at all. *Id.*

As with other items of literature filed by Petitioner in this case, there is no reason to doubt the validity of da Silva Antunes's findings. But the article does not seek to evaluate vaccine pathology, but instead to measure vaccine *effectiveness*. And the fact that it suggests that the Tdap vaccine can cause an increase of a certain kind of immune cell does not mean that the ordinary

receipt of the vaccine is likely to cause that immune cell to proliferate so much that it becomes the driver of a pathologic autoimmune process.

19. Lafdil

Lafdil appears to have been offered to link the findings from da Silva Antunes discussed above to Petitioner's theory in this case. Lafdil is a review article discussing other studies that have observed the role that Th17 T-helper cells play in the development of autoimmune liver disease. Lafdil at 250. It defines Th17 cells to be "a subset of T helper cells that play important roles in host defense against extracellular bacteria as well as in the pathogenesis of autoimmune disease." *Id.* Studies show that IL-17 is significantly elevated in a variety of chronic liver diseases, and Lafdil specifically discusses (a) a laboratory animal model form of hepatitis driven by T cells, (b) alcoholic liver disease, (c) chronic hepatitis B and C viral-induced liver infections, and (d) autoimmune liver disease. *Id.* at 251–52. The latter category arguably relates to AIH (although that term is not used in the section discussing autoimmune liver disease), but Lafdil refers mainly to studies showing increased levels of a T helper cell-associated cytokine in studies involving cirrhosis[16]—which Ms. Exum does not have—and otherwise the authors only say that the mechanisms for why these T helper cells increase in this context are unknown, along with their role in aiding pathogenesis of disease. *Id.* at 252.

20. I. Amanna et al., *Duration of Humoral Immunity to Common Viral and Vaccine Antigens,* 357 N. Eng. J. Med. 1903-15 (2007), filed as Ex. 36 (ECF No. 20-3) ("Amanna")

Amanna sought to evaluate "the issue of antibody maintenance after infection or vaccination," by reviewing 630 serum samples drawn from 45 subjects, and where the blood serum samples had been drawn regularly and "banked" over an average period of 15 years. Amanna at 1905. The study's authors looked to evaluate the temporal length of antibody maintenance (and whether the antibodies were at protective levels to fight off future disease) for a number of infections, including most of the viruses that the vaccines Petitioner received were aimed against (such as the MMR components). *Id.* at 1906). Amanna's authors determined that the antibody responses to a live viral infection were generally longer than to responses to the kind of nonreplicating protein antigens often contained in vaccines. *Id.* at 1911. But even exposure to the latter conferred some long-term benefit; the authors specifically found a comparatively-rapid decrease in tetanus-specific antibodies over a more than ten-year period. *Id.* at 1908.

Dr. Gish generally relied upon Amanna to support his contentions about the medically-acceptable timeframe in which Petitioner's post-vaccination AIH began, although he did not

[16] Cirrhosis is a condition in which an individual's liver is found to be scarred and permanently damaged, due to alcoholism or some other disease process. *Cirrhosis*, National Institute of Diabetes and Digestive and Kidney Diseases, https://www.niddk.nih.gov/health-information/liver-disease/cirrhosis#:~:text=Print%20All%20Sections-,Definition%20%26%20Facts,your%20liver%20begins%20to%20fail. (last visited on May 27, 2025).

explain in detail how it supported his contentions (which seemed more to embrace the idea that the Tdap booster caused a faster immune response due to Petitioner's prior receipt of the whole cell pertussis form of vaccine). First Gish Rep. at 23.

21. Schonberger

Schonberger is an epidemiologic study performed after the 1970s swine flu epidemic, and considers the impact of the immunization program initiated by the federal government in response. The study evaluated over 1,000 individuals who had experienced Guillain-Barré syndrome ("GBS") in the 1976–77 timeframe, comparing those who received the version of the vaccine used at that time (now nearly fifty years ago) versus those who did not, and finding that the incidence rate for GBS was higher among those who had received the vaccine. Schonberger at 109. Schonberger also made findings about the post-vaccination timeframe in which individuals developed GBS, peaking within two to three weeks but significantly declining after ten. *Id.* at 112.

Schonberger is consistently cited in the Program by virtually *any* claimant seeking to establish how the flu vaccine could cause a demyelinating autoimmune disease of the peripheral or central nervous system. Hence, *it has no direct relevance* to this case, which involves different vaccines and a different injury. Schonberger is also commonly invoked to support arguments about what constitutes a reasonable post-vaccination timeframe for development of an antibody-driven autoimmune disease - and here, Dr. Gish contended that it supported the conclusion that there was risk of a possible autoimmune, post-vaccination injury up to ten weeks after. Second Gish Rep. at 10.

22. Excerpts, Institute of Medicine, *Adverse Effects of Vaccines: Evidence and Causality*, 556-62 (Kathleen Stratton, Andrew Ford, Erin Rusch & Ellen W. Clayton, eds., 2012), filed as Ex. 45 (ECF No. 45-3) ("2012 IOM Rep.")

The 2012 IOM Report is a book-length evaluation of different risks associated with a variety of commonly-administered vaccines. Petitioner has offered a collection of excerpts from it, highlighting a few points: (a) that evaluating a potential adverse effect of vaccination involves consideration both of what the underlying natural infection does as well as the adverse event's known pathophysiology (2012 IOM Rep. at 40); (b) what the committee did in evaluating the adequacy of evidence of a particular outcome (*Id.* at 12–13); (c) that no studies (as of 2012) were considered in assessing the risk of hepatitis due to the MMR vaccine (*Id.* at 211); and (d) that case studies have associated the MMR vaccines with hepatitis, but that the mechanistic evidence of an association (whether with respect to the MMR or hepatitis A vaccine) is weak, given what is known about the natural infection (*Id.* at 212, 429–30).

B. *Respondent's Literature*[17]

1. G. Mieli-Vergani et al., *Autoimmune Hepatitis,* 4:18017 Nat. Rev. Dis. Primers 1-21 (2018), filed as Ex. A Tab 2 (ECF No. 25-2) ("Mieli-Vergani II")

The primary author of this review article is the same as Mieli-Vergani I, cited by Petitioner. Mieli-Vergani II notes some basic facts about AIH, largely consistent with Mack. However, it also makes observations about AIH that are somewhat contrary to what Petitioner gleaned from Mieli-Vergani I. In particular, Mieli-Vergani II deems the cause of AIH to be unknown (Mieli-Vergani II at 10), and in discussing potential mechanisms for its pathogenesis, like molecular mimicry, it makes no mention of measles virus (as Christen noted Mieli-Vergani I had done), focusing instead on case reports involving hepatitis infections (*Id.* at 4). But Mieli-Vergani II does propose a role for T helper cells in propagating disease, as well as the loss of immune tolerance—both of which Petitioner relies upon in her causation theory.

2. Beretta-Piccoli

Beretta-Piccoli is a case report showing the counter-risk posed by anti-malarial medicine in causing AIH. It discusses a 65-year-old woman who made yearly visits to Tanzania, and took an anti-malarial drug, Malarone (atovaquone/proguanil), on each trip. Beretta-Piccoli at 293–94. The year before the illness at issue, she had become jaundiced with an acute hepatitis following her trip, and her illness was thought to be drug-induced liver injury due to an antibiotic (although she had also been taking Malarone), although it resolved in two weeks. *Id.* at 294, 296. The following year, she returned to Tanzania and again took Malarone. *Id.* at 294. Shortly after she returned, she went to the hospital with fatigue, jaundice, and dark urine. *Id.* at 293. She reported that she had stopped taking Malarone four days earlier due to the appearance of jaundice. *Id.* at 294. After performing a liver biopsy and several lab tests, doctors tentatively diagnosed the patient with AIH. *Id.* at 295. The patient's liver inflammation took a long time to resolve, allowing a firm diagnosis only three years after presentation. *Id.* at 297. Beretta-Piccoli's authors concluded that the anti-malarial medication was the likely cause of the patient's liver pathology, but they acknowledged that a second case would be needed to confirm "the rare but potentially severe hepatotoxicity of this commonly used antimalarial prophylaxis/treatment." *Id.* at 296–97.

Respondent cited Beretta-Piccoli as evidence that anti-malarial medication can cause AIH. Crippin Rep. at 4–5. And there are similarities between this patient and Ms. Exum. Both traveled to Tanzania, and both needed prolonged therapy with corticosteroids/prednisone after falling ill. However, there are also significant factual differences. The patient in Beretta-Piccoli developed

[17] Respondent offered 38 items of literature. Exhibit List, filed January 24, 2024 (ECF No. 49). But Respondent does not bear the burden of proof in this matter, and therefore it is far less critical from a fairness standpoint that I unpack each item of literature he filed. I instead review herein only those items filed by Respondent that are most important to my analysis.

very clear signs of liver injury (jaundice, dark urine) while she was actively taking the anti-malarial medication, but Petitioner did not develop symptoms (other than fatigue) in such a short timeframe.

3. 2012 IOM Report[18]

Respondent also referenced portions of the 2012 IOM Report, in support of his contention that Petitioner had not presented sufficient evidence to support a molecular mimicry mechanism. Tr. at 184. The relevant excerpts of the 2012 IOM Report confirm that proof of homology (between different amino acid sequences in a foreign antigen and self protein structure) is insufficient to prove a molecular mimicry reaction. *See* 2012 IOM Report at 60 (deeming it "problematic" to prove autoimmunity due to molecular mimicry, and noting that homology is common in nature and usually not pathogenic). Yet Dr. Gish did not even attempt to demonstrate homology in the first place.

Additionally, the 2012 IOM Report notes that pathogenic molecular mimicry may serve as a causal explanation for an autoimmune disease if the theory is corroborated with evidence of an autoimmune attack in vivo, evidenced by proof of "local binding of antibody with activation of the complement cascade, activation of the appropriate co-stimulatory T cells signals and cytokines, and/or involvement of other pathogenic effector mechanisms in a biologically relevant tissue site." 2012 IOM Report at 71. Thus, Dr. MacGinnitie contended, Petitioner would need to show that there were antibodies or T cells that cross-reacted in her case with vaccine components, and that they were actually present in vivo. Tr. at 185. Furthermore, Petitioner would have to show that these immune cells were actually capable of mediating organ damage. *Id.* But no such evidence existed in this matter.

4. Mina

Mina is a review article discussing the significantly positive impact of receipt of the measles vaccine worldwide. *See* Mina at S11. It notes that "[m]easles immune suppression is an invisible hallmark of measles infection, predisposing to secondary infections, the major cause of measles associated mortality." Mina at S13. But "[m]easles vaccination programs have been among the greatest public health achievements" in eliminating the measles infection across the globe. *Id*. at S10. This is attributed to "direct heterologous benefits of the measles vaccines that enhance innate and adaptive immune responses." *Id*. Receipt of the measles vaccine thus not only prevents directly a subsequent measles infection, but reduces the likelihood of experiencing some secondary, opportunistic infection or illness (since the immunosuppression caused by an initial measles infection is eliminated). *Id.* at S13–15. The measles infection can have a longer-term impact on the immune system—what Mina terms "measles virus associated immune-amnesia." *Id.* at S14.

[18] *See* Ex. C Tab 2 (ECF No. 26-2)—the portions filed by Respondent are different than what Petitioner highlighted.

Respondent cited Mina to refute Petitioner's argument that the measles vaccine is capable of suppressing the immune system to any comparable pathologic degree. Tr. at 164; MacGinnitie Rep. at 8–9. As Dr. MacGinnitie explained, the measles vaccine in fact prevents the profound immune suppression caused by the measles virus (precisely what Petitioner argues in part caused her to experience AIH). Tr. at 165.

5. AC of Pediatrics

AC of Pediatrics is an annually-updated clinical manual relied upon by treaters. It acknowledges the immune-suppressive nature of the wild measles, but not the vaccine. It specifically notes that "children who have had measles have long-term blunted immune responses to other pathogens and increased mortality attributable to the known effects of the measles virus on lymphocytes." AC of Pediatrics at 503. It then discusses the importance of measles prevention (i.e. by receiving the measles vaccine). *Id.* at 503–04. Like Mina, AC of Pediatrics was cited to refute Petitioner's argument that the measles vaccine can cause clinically meaningful immune suppression, in comparison to the known impact of the underlying wild virus. Tr. at 165–66.

6. T. Safranek et al., *Reassessment of the Association between Guillain-Barre Syndrome and Receipt of Swine Influenza Vaccine in 1976-1977: Results of a Two-State Study*, 133 Am. J. of Epidemiology 940-51 (1991), filed as Ex. C-19 (ECF No. 26-19) ("Safranek").

Safranek, which involved the swine flu vaccine and GBS, found that there was an increased risk of developing GBS within six weeks of vaccination, with no increased risk after. Safranek at 940. Respondent cited Safranek to support his argument that the onset of autoimmune diseases (if vaccine-related) typically occurs no more than six weeks of vaccination (rather than up to five months, as Petitioner contended); Tr. at 176 (Dr. MacGinnitie deeming six weeks "the outer limit" for a vaccine-triggered autoimmune injury).

IV. Procedural History

As noted above, the case was initiated in 2021. Respondent filed his Rule 4(c) Report disputing Petitioner's right to compensation on August 19, 2022. Expert reports were filed through the end of 2022, and the trial was held in March 2024. I initially denied entitlement on August 29, 2024. On September 30, 2024, Petitioner filed a motion for review.

The Court granted the motion in part, and remanded the case on February 26, 2025, with instructions to elaborate on my rational for my conclusions. In particular, the Court has directed the following:

- "provide a rationale for crediting or discrediting the medical literature filed in this case; such a rationale supporting any conclusions here is necessary to

confirm that the prong one[19] legal standard was correctly applied," in order to "sufficiently articulate the legal, factual, and evidentiary bases" for my conclusion that the first prong for causation was not met (Remand Order at 43–44);

- "provide a more fulsome explanation . . . regarding (1) the probative weight of the medical literature Petitioner cited, including Petitioner's case reports involving relevant vaccines and vaccine antigens, (2) his rationale for accepting or rejecting that evidence; and, as necessary, (3) the extent to which he is crediting each expert's testimony; and (4) his rationale for doing so" (Remand Order at 44);

- "to the extent that, on remand, . . . analysis of prong one affects [my] findings pertaining to Petitioner's prong two logical sequence of cause and effect, [I] should revise and explain those findings accordingly" (Remand Order at 47); and

- "(1) revise [my] findings on prong two, only to the extent that [my] prong one analysis on remand affects those findings, (2) revise [my] analysis pertaining to alternative causes, insofar as [I] improperly required Petitioner to "persuasively limit or exclude *all of them*," and, importantly, (3) fully describe [my] rationale for all of his prong two conclusions in the remand decision" (Remand Order at 50–51).

V. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[20] There is no Table injury for AIH, so Petitioner can only assert a causation-in-fact claim.

[19] Prong references are to the Federal Circuit's decision that established the causation standard applied in Vaccine Program cases—*Althen v. Sec'y of Health and Hum. Servs.,* 418 F.3d 1274, 1278 (Fed. Cir. 2005).

[20] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121,

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health and Hum. Servs.,* 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

Each *Althen* prong requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Distinguishing between "preponderant evidence" and "medical certainty" is important because special masters must take care not to impose an evidentiary burden that is too

124 (2003), *aff'd* 104 F. App'x. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

high. *Bunting v. Sec'y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed.Cir.1991) ("The standard of proof required by the [Vaccine] Act is simple preponderance of evidence; not scientific certainty.... [I]t is not plaintiff's burden to disprove every possible ground of causation suggested by defendant nor must the findings of the court meet the standards of the laboratorian.") (citations and internal quotation marks omitted).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Demore v. Sec'y of Health & Hum. Servs.*, No. 20-1265V, 2024 WL 4542934 (Fed. Cl. Spec. Mstr. Sept. 26, 2024), *aff'd*, No. 20-1265V, 2025 WL 868902, at *4 (Fed. Cl. Mar. 20, 2025) (rejecting the argument that a petitioner's burden is to prove that a causation theory is *plausible* and instead requiring petitioner to prove the theory by a preponderance of the evidence) (emphasis added). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("*Snyder II*") ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in

its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of

expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder II*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

ANALYSIS

I. Treatment of AIH in Prior Cases

The parties agree that Ms. Exum was properly diagnosed with AIH. But even though this is undisputed, some brief discussion of AIH's characteristics will help in the analysis of causation in this matter.

According to Mack, AIH occurs when "a break in self-tolerance to hepatocyte autoantigens initiates immunological responses causing progressive hepatic necroinflammation and fibrogenesis." Mack at 675, Figure 1. Mack deems AIH to be a "complex genetic disease that requires interplay among genetic, epigenetic, immunologic, and environmental factors." *Id.* at 674. It allows that environmental factors like "viral infections or xenophobic disorders" can defeat immune tolerance against autoantigens in genetically-susceptible persons, although it does not mention vaccines. *Id.* Nonspecific symptoms like fatigue and arthralgias are common to its

presentation. *Id.* at 680; Crippin Rep. at 3, 6. An AIH diagnosis can be confirmed by liver tissue biopsy. Crippin Rep. at 6.

The primary articles regarding AIH offered by either side do not appear to embrace the concept that vaccines might in rare cases cause it. At best, they propose an immune-mediated process for how it unfolds. Mack, for example, describes the pathogenesis of AIH as involving a breaking of immune tolerance mediated by a variety of T cells, while cross-reactive autoantibodies (here, presumably incurred due to vaccination) are generated. Mack at 674–76. This is largely consistent with Petitioner's proposed mechanistic theory. Mack also sets forth a number of specific autoantibodies associated with AIH (although Petitioner has never been shown to have possessed any). *Id.* at 676–78. But Mack makes no mention of the *measles virus* as a potential cause of AIH—and its discussion of vaccination-related risks is limited to the context of existing AIH patients receiving immunosuppressive treatments, as opposed to individuals not already experiencing AIH. *Id.* at 686.[21] Indeed—immunosuppression is a *favored treatment* of AIH. Mieli-Vergani II at 1, 2, 11 ("AIH should always be treated with immunosuppressive drugs with very few exceptions").

The Vaccine Program has previously considered whether certain covered vaccines can cause AIH—but the trend seems to be against a finding of causation. *See, e.g.*, *Porter v. Sec'y of Health & Hum. Servs.,* 663 F.3d 1242 (Fed. Cir. 2011) (reversing Court's rejection of special master determination that hepatitis B vaccine was not shown to cause AIH; special master's decision was reasonably based on credibility determinations about the competing expert opinions, as well as other record evidence). In addition, most such prior claims logically focused on the hepatitis A or B vaccines, since their viral cognates are understood to lead to hepatitis (and indeed, the bulk of the case reports filed in this case better support *that* kind of causation case than what is advanced here).

There are comparatively fewer cases involving the MMR or Tdap vaccines—but those that exist are not favorable to Petitioner. *See, e.g.*, *Rivas v. Sec'y of Health & Hum. Servs.*, No. 21-1683V, 2025 WL 551570, at *2 (Fed. Cl. Spec. Mstr. Jan. 24, 2025) ("[P]etitioner has not carried her burden of presenting a minimally persuasive case that the Tdap vaccine and hepatitis A and B vaccines can cause and/or worsen autoimmune hepatitis").

II. Petitioner Has Not Met her Burden of Proof under *Althen*

The failure to establish even one of the three *Althen* prongs in the context of a causation-in-fact claim is sufficient basis for a claim's dismissal. *Dobrydnev v. Sec'y of Health & Hum. Servs*., 566 Fed. Appx. 976, 980 (Fed. Cir. 2014). Upon remand, I once again determine that Petitioner did not preponderantly establish causation—at least with respect to the first two *Althen*

[21] Mack actually notes the importance of vaccinating "[p]atients unprotected against infection with hepatitis A virus (HAV) and hepatitis B virus)(HBC)" before they undergo immunosuppressive treatment for AIH. Mack at 686.

prongs. (But although the Remand Order affirms the fact that a special master is not compelled to perform analysis of each prong individually, if at least one is found not to have been satisfied (Remand Order at 51), I am including herein my analysis of all three prongs).

A. *Prong One*

Petitioner contends that the MMR and Tdap vaccines—alone or in combination—more likely than not can cause AIH. Some elements of the theory are wholly noncontroversial, since they begin with propositions rooted in what is known about the pathogenesis of AIH. Thus, AIH likely occurs due to some combination of individual susceptibility and an environmental trigger. But the theory *in its totality* lacks sufficient preponderant support, even if some individually-reliable items of literature have been offered in this case. In order to fully meet the Court's remand mandate, I have explicitly broken down each element of my reasoning for why I find causation has not been demonstrated.

1. Petitioner has Not Preponderantly Demonstrated a Link Between the Measles Component of the MMR Vaccine and AIH

The primary thrust of Petitioner's causation argument was that the measles component of the MMR vaccine has the capacity to suppress the immune response, opening the door thereafter to AIH (with the Tdap vaccine possibly playing a secondary role). There are several flaws with the theory.

To an overarching degree, Petitioner's theory is rooted in a false equivalence between the immunologic impact of the measles wild virus versus the vaccine.[22] Certainly Program claimants often base their theories on the impact a vaccine's wild viral or bacterial counterpart can have from a pathologic standpoint (since that opens the door to the possibility that a vaccine based on that same wild virus could achieve a comparable effect). And there is reliable evidence that the wild measles *infection* can cause pathologic harm due to immune suppression—even after the virus has been successfully treated and/or cleared.

But the measles wild virus has not been shown to be directly associated with AIH (and instances of liver disease or even AIH associated with hepatitis infections of any kind cannot inform the analysis herein). And it cannot be disputed that the danger posed by the wild measles virus is *far greater* than the vaccine, all things being equal. *See, e.g.*, McMahon at 635 (measles infections "complications, often due to [virus] induced immunosuppression, occur in up to 30% of cases, and can result in pneumonia, encephalitis, and, in rare cases, death"). Accordingly, it is not facially compelling for Petitioner to assume that vaccination *per se* involves a risk comparable to the impact of the wild measles infection.

[22] *See, e.g.*, Tr. at 37 (Dr. Gish responding affirmatively to the question, "[w]ould it be expected that Mrs. Exum suffered a mild vaccine strain measles infection from the administration of her vaccine?").

More specifically, however, the contention that the measles vaccine is capable of pathologic immune suppression has not been preponderantly established—even if *some* of the items offered to support this concept have scientific reliability. Both Munyer and Nanan support the narrow proposition that the measles vaccine has been experimentally observed to produce some depression of T cell lymphocyte function—but that is where their findings end. Neither article stands for the conclusion that the studied patients experienced *meaningful* immune suppression, such that any disease became more likely after receipt of the vaccine (let alone AIH). *See, e.g.*, Munyer at 76. The dated quality of these two items of literature further reduces the weight to be given to them, since they were never followed by confirmatory studies that substantiate the importance of their findings. If it has been known for over twenty years that a measles virus-containing vaccine could produce a dangerous degree of immune suppression, where are the subsequent studies evaluating or testing this purported risk?

Thus, the concept that some fairly-old studies produced some evidence of *possible* vaccine-related suppression needed to be linked to *other* evidence fleshing out the proposition—not necessarily specific to AIH, but at least to some comparable autoimmune injury. But the evidence Petitioner has offered does not fill these holes in the theory very well. Christen, for example, only briefly mentions the measles vaccine, but notes that evidence connecting it to AIH was not corroborated over time. Christen at 43. Christen similarly mentions a case series article never filed in this case (Vento), and which observed only a *temporal* association between measles and AIH. And it references Mieli-Vergani I as the ultimately-uncorroborated measles virus-AIH connection—while the same primary author (in the more recently-published Mieli-Vergani II) made no mention at all of measles virus as a putative AIH trigger (suggesting that in the 30 years that passed between the two articles, the measles trigger hypothesis was no longer considered important).

Robertson is fairly weak support for the proposed measles-AIH link. It may well show (in a small sample) that a preexisting measles infection might be associated with an AIH-like condition. Yet (and in addition to the fact, noted in Christen, that Mieli-Vergani I did not corroborate its potential findings—and that the illness considered may not be on all fours with AIH) it says little about the impact of receipt of a measles-containing vaccine. I therefore do not find that even the nascent *possibility* outlined in Nanan and Munyer has been subsequently confirmed or carried forward—meaning in turn that this aspect of Dr. Gish's theory lacks sufficient reliable support to corroborate it.

I also emphasize that the argument that the measles vaccine component can cause pathologic immune suppression is hardly new to the Program. In fact, it was a central pillar of the causation theory advanced in the Omnibus Autism Proceeding (the "OAP") over 15 years ago, about the alleged capacity of the MMR vaccine to cause autism. But (after a matter characterized

by a lengthy and laborious degree of fact-finding unheard of in the Program, before or since)[23] the idea that pathologic immune suppression could be caused by this vaccine was *firmly rejected*. *See, e.g.*, *Snyder v. Sec'y of Health & Hum. Servs.*, No. 01-162V, 2009 WL 332044, at *104 (Fed. Cl. Spec. Mstr. Feb. 12, 2009) ("petitioners have failed to demonstrate that the MMR vaccine causes immunosuppression. There is no evidence that children receiving the vaccine have higher rates of infection in the months after vaccination than children who do not receive the vaccine"), *mot. for review den'd,* 88 Fed. Cl. 706 (2009).

Notably, Nanan and Munyer were published before *Snyder's* issuance—again raising the question as to why, if they stand as reliable and persuasive evidence of MMR vaccine-caused immune suppression, there are not any more recent follow-up studies corroborative of their findings. And subsequent decisions considering the same proposition (admittedly, also in the context of an autism injury claim) have rejected the theory again, even where such articles were expressly invoked in support. *See, e.g.*, *Reed v. Sec'y of Health & Hum. Servs.*, No. 08-650V, 2018 WL 6844458, at *47 (Fed. Cl. Spec. Mstr. Dec. 4, 2018) (rejecting contention that MMR vaccine can cause clinically meaningful immunosuppression resulting in autism, and discussing Munyer's limits as persuasive evidence to that point).

Petitioner's immunosuppression argument is further weakened by Respondent's counterevidence, which supports the conclusion that the measles vaccine actually makes immune suppression far *less* likely—simply by reducing the possibility of a measles infection in the first place. *See e.g.*, Mina at S10 (noting that measles vaccine is correlated with large reductions in childhood mortality because direct heterologous benefits of the vaccine enhance innate and adaptive immune responses). And there is a contradictory quality to arguing that immune *suppression* would cause a disease reaction that is the product of an *uncontrolled* immune response—since autoimmunity involves an unregulated attack by the immune system against self. In fact, as Dr. Crippin noted, immunosuppressive treatments are used for AIH, and Petitioner herself received them (Crippin Rep. at 3). How does measles-induced immunosuppression lead to a disease that *itself* is treated by immune suppressive drugs? Mieli-Vergani II at 1, 2, 11. This contradiction is unanswered by Dr. Gish.

Otherwise, the evidence of a direct MMR *vaccine*-AIH connection is largely absent.[24] It is limited to a single case report, Saliba (with Veerappan providing one additional case involving the

[23] *See Hooker v. Sec'y of Health & Hum. Servs.,* No. 02-472V, 2017 WL 3033940, at *4–6 (Fed. Cl. Spec. Mstr. Apr. 11, 2017), for a summary of the OAP process. The work performed by the three special masters who jointly heard evidence in the OAP, and then wrote the formidably-long entitlement decisions thereafter, was exemplary—and thus findings made therein that have relevance to other kinds of Vaccine Act claims *deserve considerable weight*, even if they do not control the outcome herein.

[24] My observation that this evidence is lacking is not the same as *requiring it*. I am simply noting that the evidence of a more direct vaccine-AIH association is not evident (underscoring the degree to which Petitioner must rely on more indirect and circumstantial proof—and here, that proof too proves inadequate).

MMR, but too many other additional vaccines to separate out possible triggers). This is not enough of a link to remedy the other deficiencies in this aspect of Petitioner's causation theory.

I thus do not find on this record that it is preponderantly likely that the MMR vaccine can cause sufficient levels of immune suppression to have a clinically-meaningful impact—such that it could establish conditions for an autoimmune disease like AIH.

2. Evidence About the Putative Role of the Tdap Vaccine in Causing AIH is Weak or Lacking

Dr. Gish devoted less time at trial to explaining his theory for how the Tdap vaccine could play into the proposed pathogenesis for AIH. But this aspect of the overall theory made assumptions about the impact of the Tdap vaccine (and its promotion of inflammation through stimulation of T-helper cells) that arise more from supposition than independent evidence.

Petitioner contends that an upregulation of T helper cells due to receipt of a Tdap booster could in turn cause pro-inflammatory cytokines secreted by those T helper cells to encourage an autoimmune disease process. First Gish Rep. at 21–22. The limited evidence that he offered for this was unconvincing. In van Gemeren, for example, a woman developed AIH after receiving multiple vaccines, including the Tdap vaccine. van Gemeren at 19. But the authors of this case report did not include *any* discussion of the Tdap vaccine. *Id.* Instead, they narrowed in on the hepatitis A vaccine as a potential trigger. *Id.* at 21–22.

da Silva Antunes admittedly provided some evidence relevant to this argument, finding that people previously vaccinated with the whole cell pertussis vaccine produce more IL17 cytokines after booster vaccination (for the subset of the sample that previously received a whole cell pertussis version of the vaccine—presumably true of Petitioner as well). da Silva Antunes at 3–4. But does this mean that the levels of cytokines generated in reaction would be enough to be pathogenic—if not in the specific context of AIH, then with respect to *any* autoimmune disease? da Silva Antunes does not say. And its more limited observation was rooted in an evaluation of the comparative immunologic value of acellular versus whole-cell pertussis - not the pathogenic potential of the Tdap vaccine. da Silva Antunes at 3855–56. The article includes no discussion of AIH. And Dr. MacGinnitie persuasively also pointed out that the sample size of the da Silva Antunes study was limited, that it did not observe large increases in the production of these cytokine-generating helper cells, and otherwise that these T cells themselves are not considered per se pathogenic in any event. First MacGinnitie Rep. at 9–10.

Lafdil does not bridge this evidentiary gap sufficiently. It does observe that T-helper cells play *some* role in the development of autoimmune liver disease—in particular through encouragement of a kind of pro-inflammatory cytokine associated with different kinds of liver illnesses—some of which are analogous to AIH. Lafdil at 250, 251–52. Again, however—does

this lead to the conclusion that receipt of a Tdap booster is likely to raise these cytokines to pathogenic levels, *and* thereupon spur on AIH? Lafdil does not so conclude, and it is speculative to do so here. Petitioner has only shown that there is scientific support for the conclusion that this kind of T helper cell is involved in an AIH-like disease process. But that is not the same as the determination that vaccination likely *leads* to the disease process. There are too many missing links in the causation chain to so conclude.

Overall, this element of the causation theory reflects Petitioner's continued effort to transmute narrow, scientifically-reliable facts (derived from limited but methodologically reasonable studies) into a basis for pathology which *itself* is ultimately speculative. Even if a Tdap booster results in the production of certain T cells that could be associated with autoimmune disease, this does not mean they *drive or initiate* that disease—or that they are upregulated by vaccination in amounts sufficient to cause disease. And Petitioner's showing, in the absence of other evidence implicating the Tdap vaccine (or its viral/bacterial components) to AIH, is even more faint in supporting causation.

3. Insufficient Evidence Was Offered to Establish The Risk of Receiving Two Vaccines at Once

Dr. Gish argues that the administration of both vaccines at the same time raised the risk of an aberrant response. Tr. at 51, 63. But this is a *barely* plausible contention that lacked corroboration from materials filed in this case. No studies were offered that evaluated via a trustworthy methodologic experiment the impact of receiving two vaccines at the same time – in the context of any autoimmune disease. At most, some case reports were filed in which at least one of the relevant vaccines was administered. *See, e.g.*, Veerappan, van Gemeren. While the authors of Veerappan suggest that AIH may be a complication in patients who receive multiple vaccinations simultaneously, they do not explain why. Veerappan at 212. And in Veerappan, the patient received *four vaccines simultaneously*, as opposed to Petitioner's two. (Otherwise, and as discussed in greater detail below, there is no evidence in the contemporaneous medical record at all of any suspect, close-in-time reaction to the vaccines Petitioner received on August 20, 2018—further distinguishing the present case from Veerappan).

4. Dr. Gish's Molecular Mimicry Mechanism for Driving AIH Due to Vaccination Was Inadequately Corroborated

Admittedly, Petitioner in this case did not primarily rely on a theory that antigenic similarity between components of either vaccine she received and protein components in the liver caused an autoimmune cross-reaction, resulting in AIH. Dr. Gish, however, did invoke the concept as a reliable mechanistic explanation for autoimmune disease. *See, e.g.*, First Gish Rep. at 10, 17,

20–21. But he did not substantiate it adequately either, such that I could find it might stand as a reliable mechanism explaining how the two relevant vaccines, alone or in combination, could lead to AIH.

Some autoimmune diseases can be mediated by mistaken self-attacks propagated by antibodies generated in response to a foreign antigen that (due to molecular and/or structural similarities with a self-antigen protein) then cross-react with the self tissue, often in a chronic/persistent manner. *See*, *e.g.*, *Bielak v. Sec'y of Health & Hum. Servs.*, No. 18-761V, 2023 WL 35509, at *33 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) ("[m]olecular mimicry is predominantly driven by B cell activity, occurring when antibodies are produced in response to antigenic components of the vaccine—but which (due to mimicry between the presenting vaccine antigens and self-tissues) cause harmful cross-reactions, by mistakenly attacking the self antigens").

Because vaccines are usually designed to encourage an adaptive response to a foreign antigen specifically by "teaching" the immune response to create antibodies to attack the antigen in future exposures, the mechanism of molecular mimicry fits well with theories that the immune response to vaccination could become pathologic. But litigants who invoke molecular mimicry to explain how a vaccine could have triggered an autoimmune disease commonly attempt to show at a minimum (a) what homology might exist between a vaccine's antigens and a self-structure relevant to the situs of harm (here the liver), and (b) what evidence exists that the vaccine would cause the production of the likely pathogenic antibodies. *See K.A. v. Sec'y of Health & Hum. Servs.*, No. 16-989V, 2022 WL 20213037, at *9 (Fed. Cl. Spec. Mstr. Apr. 18, 2022), *mot. for review den'd*, 164 Fed. Cl. 98 (2022), *aff'd*, 2024 WL 2012526 (Fed. Cir. May 7, 2024) ("[b]ecause homology is common in nature (given the total limited number of amino acids that constitute proteins), it is important to focus on homology specific to the "disease-related" situs for the cross-reactive attack").

Here, however, Dr. Gish has not made a homology showing—which, as Dr. MacGinnitie noted, is by itself not enough evidence to conclude an autoimmune process linked to vaccination has been established. He also did not otherwise offer evidence suggesting that the vaccines or their wild viral counterparts are associated with any antibodies thought to drive AIH or be involved in its pathogenesis. There is thus very little evidence in this record that would support molecular mimicry due to vaccination as a mechanistic explanation for AIH.

In reaching my conclusion about the strength and applicability of molecular mimicry as a mechanism in this case, I acknowledge that petitioners need not offer any mechanism at all in proving causation. *Andreu*, 569 F.3d at 1378–79**.** But what does this mean, in a case where the Petitioner's expert *did* offer *several* possible mechanisms? Is the special master obligated to avert his eyes from the strength of the evidence at issue? Must he simply assume a briefly-invoked mechanism reasonable, without consideration of the weaknesses noted in it by Respondent?

Relevant and controlling case law says otherwise. *Howard v. Sec'y of Health & Hum. Servs.*, No. 16-1592V, 2022 WL 4869354, at *24 ("[o]f course, petitioners are never required to

establish mechanism—but they often attempt to do so, and therefore it is reasonable to evaluate their success in the effort") (Fed. Cl. Spec. Mstr. Aug. 31, 2022), *mot. for review den'd,* 2023 WL 4117370 (Fed. Cl. May 18, 2023), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024). I may evaluate whether a proposed pathologic mechanism has been evidentiarily supported—and this one has not. Claimants cannot both invoke a possible mechanism to flesh out their causation theory, but then evade its deficiencies by objecting they were never obligated to prove it in the first place.

5. Case Reports are Weak Causation Evidence, and Those Offered Were Largely Unhelpful to Petitioner

Petitioner offers a number of case reports in support of her causation argument. *See* Saliba, Sasaki, Perumalswami, Subramanian, van Gemeren, Berry, Veerappan. At the outset, however, it is plain that several of them (Berry, Perumalswami, Sasaki) do not involve the Tdap or MMR vaccines (or even comparable wild virus infections). Berry, Perumalswami, and van Gemeren involve the hepatitis A and/or B vaccine; Subramanian a hepatitis A wild infection; and Sasaki the flu vaccine. This fundamental fact distinction greatly saps such case reports of evidentiary value in the context of this case. *See Herms v. Sec'y of Health & Hum. Servs.*, No. 19-70V, 2024 WL 1340669, at *21 (Fed. Cl. Spec. Mstr. Mar. 4, 2024) ("Petitioner does not explain how data from other unrelated vaccines could be extrapolated to the vaccines at issue here and accordingly, the data is not persuasive"), *mot. for review den'd*, 173 Fed. Cl. 1 (2024); *see also Deshler v. Sec'y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162, at *19–21 (Fed. Cl. Spec. Mstr. July 1, 2020) (declining to attribute case reports on the flu vaccine to the causation potential of pneumococcal vaccines).

Saliba is the most factually-relevant case report, since it involves a patient who developed hepatitis after receipt of the MMR vaccine. Saliba at 379. But the patient in Saliba experienced *acute* hepatitis, which is distinguishable from AIH, a *chronic* condition. Additionally, the patient in Saliba developed obvious symptoms of liver disease within two weeks of vaccination, while Petitioner's first confirmed symptom (elevated liver enzymes) appeared two months post-vaccination. It is thus factually distinguishable in important respects.

In a larger sense, case reports are of limited value in determining causation—even when they are factually relevant. Special masters have repeatedly observed that case reports present only a temporal sequence of events, and thus stand as very thin evidence of causation. *Demore v. Sec'y of Health & Hum. Servs.*, No. 20-1265V, 2024 WL 4542934, at *7 (Fed. Cl. Sept. 26, 2024), *aff'd*, No. 20-1265V, 2025 WL 868902 (Fed. Cl. Mar. 20, 2025); *see also Campbell v. Sec'y of Health & Hum. Servs.*, 97 Fed. Cl. 650, 668 (2011) ("'[c]ase reports do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value,' even if they should receive some weight").

Dr. Gish devoted much of his supplemental expert report to attempting to defend case reports, especially given the rarity of vaccine injuries. *See generally* Second Gish Rep. at 1–4. But his reasoning rings hollow. A case report may be the place to *start* a causation inquiry, but that effort must then build upon other evidence that suggests the temporal/coincidental observation of post-vaccination injury is scientifically meaningful. And here, the paucity of case reports specific to the vaccines at issue and AIH do not appreciably add to the total picture. Dr. Gish reasons that case reports suggest a one-time occurrence might repeat elsewhere (Second Gish Rep. at 3 ("if something did happen in another person, then logically the event can happen in this patient")—but this kind of logic does not pass muster in the Vaccine Program when uncorroborated with other evidence.

6. Other Proposed Mechanisms Were Inadequately Developed

Petitioner's causation showing included a number of additional independent theories or mechanisms that were only briefly touched upon, but never substantiated into something sufficient to be deemed preponderantly established. These contentions accordingly merit even less weight than the better fleshed-out aspects of the causation theory discussed above, like the proposed impact of measles vaccine-associated immune suppression.

For example, as evidence for the contention that the measles vaccine component could literally "infect" certain immune cells, Petitioner referenced Rennick. But as I have noted above, an article like Rennick has more to say about the function of the measles vaccine from an immunologic standpoint than it does about the vaccine's allegedly-pathogenic capacity (which is what is at issue in this case). Its observation about the way the vaccine's antigens are taken up by immune cells cannot be alone stretched into a finding that this in turn increases the likelihood the vaccine will, at some later time, unexpectedly cause measles-associated immune suppression of some kind. Thus, Rennick stands as another instance in which Petitioner seeks to take anodyne scientific findings about vaccine performance and speculatively recast them into proof of pathology.

Petitioner proposed bystander activation of nonspecific immune cells (and/or suppression leading to expansion of these immune cells) as another way AIH might occur. First Gish Rep. at 9, 10; Tr. at 42, 58. But there was inadequate evidence supporting this mechanism as likely causal of AIH due to vaccination.[25] Dr. MacGinnitie persuasively explained that usually bystander activation would only be thought to occur in the presence of active inflammation due to an infectious process (which was never shown to have happened in this case). Tr. at 158–60. And

[25] In fact, articles like Mack did not even mention bystander activation as an understood mechanism for AIH (Tr. at 160)—although Dr. MacGinnitie admitted on cross-examination that Mina mentioned bystander activation as something a vaccine could encourage—blunting this contention somewhat.

articles like Benn are focused on the positive, secondary effects of this kind of nonspecific immune stimulation, rather than supporting the contention that vaccine-associated bystander activation is harmful. Benn at 432, 436–37.

Dr. Gish also spoke generally of immune tolerance being broken by a T cell-driven process, resulting in harm to liver cells. Tr. at 53–54; Petitioner's Post-Hearing Brief, filed May 31, 2024 (ECF No. 73) ("Br.") at 9. This is consistent with how AIH likely unfolds—but a description of the pathogenesis of the disease in question does not also establish causation, if not yoked to sufficient reliable proof to conclude the vaccine can be an aspect of the disease process. I have already observed above that explanations like measles-associated immunosuppression, or Tdap booster-driven T helper cell increases, do not "work." And certainly no other, more direct evidence was offered to show how these vaccines would break immune tolerance. Petitioner could not simply take a truism about the illness in question and then turn that into evidence of vaccine-instigated pathology, without better and more persuasive proof.

7. Respondent's Experts Were More Persuasive Than Dr. Gish on the Question of Vaccine Causation

Another factor bearing on my prong one determination is my assessment of the experts, who I saw and heard live at hearing. Dr. Gish was certainly *qualified* to offer an opinion in this case as a general matter, given his expertise in hepatology. But he has no specific expertise studying AIH's pathogenesis (even if he has a background in the treatment of liver-associated diseases, and has some reasonable insights gleaned from that experience). In addition, he was matched against another, equally-qualified hepatologist (Dr. Crippin) who *also* reasonably relied on his own experience in questioning causation. Worse, Dr. Gish lacks demonstrated expertise in the field of immunology—and the opinions he offered on this subject were persuasively rebutted by Dr. MacGinnitie—*the sole immunologist* who testified herein. I therefore have given Dr. Gish's generalized and somewhat broad contentions about the alleged immune process that could lead to AIH post-vaccination less weight than the Respondent's experts' testimony.

This is not a case where I deemed an expert to have lacked core "credibility."[26] But weighing of expert testimony is not limited to determining bare-bones questions of personal honesty or truthfulness. Nor does Program law *ever* compel me to accept an expert's *ipse dixit*. *Snyder II*, 88 Fed. Cl. at 743. While Dr. Gish *presented* an opinion favorable to Petitioner, *that was not the end of my evaluation of it*—and as discussed herein, I deem it ultimately wanting, since

[26] I do, however, share the opinion offered by Dr. MacGinnitie that it was "difficult to follow a logical, step by step theory of causation in Dr. Gish's Report," which was excessively wordy, meandering, and unclear in the theory it embraced. MacGinnitie Rep. at 6. His enunciation of that theory at hearing was not much more illuminating—and although I have attempted to summarize his views in a more ordered fashion, it has not been easy (and this also detracts from the theory's persuasiveness).

it was unsupported by sufficient corroborating evidence. Dr. Gish too often tried to leverage things known about AIH into proof of vaccine pathology, without sufficient connective evidence; exaggerated the impacts of the measles vaccine into a disease process; and relied on older studies that have not been subsequently shown to have the same significance they might once have been thought to possess. Dr. Gish's theory did not add up, independent of his expertise as a hepatologist, and even if some of its components were individually reliable.

In summary: Petitioner was unable to establish that the Tdap vaccine and/or MMR vaccine can likely cause autoimmune hepatitis. I make this finding based on an in-depth review of all the evidence presented by both parties. Petitioner has not provided enough reliable evidence that the MMR vaccine causes clinically significant immunosuppression leading to an autoimmune disease like AIH. For these reasons, I find that the first *Althen* prong is unsatisfied.

B. *Althen Prong Two*

Under the second *Althen* prong, a petitioner must prove actual causation—that the vaccine(s) at issue "did cause" the alleged injury/illness—by a preponderance of the evidence. *Boatmon*, 941 F.3d at 1355.

The evidence in the record for this matter *does not* so preponderate. I mostly base this determination on the absence of objective record proof from which it could be inferred that Petitioner's August 20, 2018 vaccinations caused any aberrant reaction. Thus, Petitioner's medical records contemporaneous to her vaccination date are devoid of evidence that she experienced any close-in-time vaccine reaction. In addition, she never tested positive for inflammation biomarkers that might corroborate that she was experiencing some kind of inflammatory event *before* her liver test values became elevated. Petitioner was not shown to have possessed an active measles infection (key to Dr. Gish's opinion about suppression), and was not demonstrated to be experiencing any form of hepatitis infection, or even any *other* opportunistic infectious process that could be thought to have occurred in the wake of the alleged measles vaccine-caused immune suppression (and thus might demonstrate indirectly that she was then suffering from immune suppression). I also have not ascertained any instance where any of Petitioner's treaters proposed the vaccinations explained her AIH (including Drs. Oloruntoba or Wu).

What remains is the fact that Petitioner experienced some post-travel nonspecific symptoms which did not immediately merit treatment (and which arguably were consistent with her pre-vaccination health). While these concerns (in particular, fatigue) seem likely related to Petitioner's subsequently-diagnosed AIH, they are not enough of a basis to conclude that the two vaccines Petitioner received "did cause" her AIH. Petitioner received the two vaccines, traveled abroad, then a few weeks later felt fatigue, and then inadvertently was found to possess elevated LFTs. To deem this sufficient to establish "a logical sequence of cause and effect" as required for

the "did cause" prong would be to elevate the temporal relationship between vaccination and injury (here, already somewhat attenuated) into compelling proof of causation. *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992) ("[a] proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury").[27]

It is also very important to give weight to the circumstances in which Petitioner received these vaccines—for her vaccinations did not occur in a vacuum. While Dr. Gish did observe the absence of some risk factors for AIH relevant to Petitioner (for example, she had not tested positive for a hepatitis viral infection), he did not persuasively account for the risk factors that *were* present—even though he acknowledged the relevance of this kind of evidence to causation. Tr. at 20 (Dr. Gish testifying that reviewing an individual's medication history is "really key" to determining what may have caused AIH). Petitioner's recent post-vaccination foreign travel (which clearly could have exposed her to any number of possible pathogens), plus her acknowledged receipt of anti-malarial medications and supplements, were all potential causal factors.

The anti-malarial medication Petitioner received is an example of the kind of confounding factor undermining vaccines as causal in this case. It is well-established that medication toxicity is a risk factor for liver dysfunction. *See e.g.*, Tr. at 86 (Dr. Gish acknowledging the existence of drug-induced liver disease). And in Beretta-Piccoli, a patient developed long-term autoimmune hepatitis (comparable to AIH) while taking the same anti-malarial medication as Petitioner. Beretta-Piccoli at 293–95. The fact that this case report patient developed hepatitis on *two* separate occasions while taking this medication led the authors to conclude that the medication was "likely" the cause of the patient's liver pathology. *Id.* at 296. And the patient in Beretta-Piccoli stopped taking the anti-malaria meds four days before she presented at the hospital, but still developed AIH. Beretta-Piccoli at 294.

Admittedly, it cannot be concluded from this record that the anti-malarial medication *likely* caused Petitioner's AIH. Dr. Gish reasonably noted that this medication typically does not result in long-term autoimmune disease, and/or should cause harm closer-in-time to when the medication is being taken. He similarly pointed out some factual differences in the form of hepatitis at issue, as well as the relevance of when the medication is taken in comparison to onset. And I am reluctant to give great weight to a case report *of any kind* – whether offered by Respondent *or* Petitioner.[28]

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[27] For this same reason, I do not give weight to the *pre-vaccination* absence of evidence of liver disease or hepatitis as proof of the vaccine's role in causing what manifested after. The fact a claimant was not sick before the vaccination, but sick after, is simply another form of *post hoc ergo propter hoc* reasoning that the Program rejects.

[28] Of course, if the "signal" of otherwise-factually distinguishable case reports like Saliba should be viewed as credible evidence of causation—as Petitioner urges—then there is no reason to downplay the findings of Beretta-Piccoli simply because Dr. Gish observes some factual distinctions between that case and the present facts. The same distinctions are true for Saliba, which involved acute hepatitis and a short onset—and as the adage goes, "sauce for the goose is sauce for the gander." I also note that Beretta-Piccoli is considerably more substantive in its analysis than Saliba – the most on-point case report offered by Petitioner, but which takes the form of a one-page "letter to the editor," and includes far less consideration of causality.

But this medication is clearly something that had a demonstrated capacity to increase Petitioner's possible hepatitis risk, and she received it *after* vaccination (and hence closer in time to her possible onset).

I am not, on the basis of this record, able to determine what did likely cause Petitioner's AIH—and I do not find Respondent preponderantly established a different trigger. But entitlement is not properly granted simply because Respondent cannot "prove" an alternative to vaccination as the cause. *Winkler v. Sec'y of Health & Hum. Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) ("the failure to prove an alternate cause does not obviate the need for proof of causation by the vaccine"). *Only* when a claimant has met his initial prima facie case is Respondent even burdened with proving a "factor unrelated." Section 13(a)(1)(B); *Althen*, 418 F.3d at 1278. And I have not found the burden should be, or was, shifted here.[29]

The Court's Remand Order accurately observed that there can be a "fine line" between noting the existence of evidence of alternative explanations for a claimant's injury, and actually requiring the Petitioner to *disprove* them. Remand Order at 50 (citing *Sharpe v. Sec'y of Health & Hum. Servs.*, 964 F.3d 1072 (Fed. Cir. 2020)). And petitioners are never obligated formally to disprove alternative causal factors. *Walther v. Sec'y of Health & Hum. Servs.*, 485 F.3d 1146, 1149–50 (Fed. Cir. 2007) (citing *Pafford*, 451 F.3d at 1359).

But it is fully appropriate for special masters to evaluate and weigh record evidence of contrary possible explanations for an injury in assessing a claimant's prong two success. *Winkler*, 88 F.4th at 963 ("contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law"); *Stone v. Sec'y of Health & Hum. Servs.*, 676 F.3d 1373, 1380 (Fed. Cir. 2012) ("[t]he special master is entitled to consider the record as a whole...and no evidence should be embargoed from the special master's consideration simply because it is also relevant to another inquiry under the statute"). This weighing reasonably includes review of evidence about the Petitioner's health or other circumstances bearing on their illness.[30]

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[29] I have conducted the burden-shifted "factor unrelated" analysis in cases where a petitioner carried his initial burden—and the outcome thereafter has varied, depending upon the record and expert evidence offered to interpret it. *Compare White v. Sec'y of Health & Hum. Servs.,* No. 20-1319V, 2023 WL 4204568 (Fed. Cl. Spec. Mstr. June 2, 2023) (infection caused petitioner's GBS as factor unrelated, even though prima facie Table claim of GBS after receipt of flu vaccine was met), *mot. for review den'd,* 168 Fed. Cl. 660 (2023), *appeal docketed,* No. 2024-1372 (Fed. Cir. Jan. 23, 2024), *with Taylor v. Sec'y of Health & Hum. Servs.,* No. 22-335V, 2025 WL 1234906 (Fed. Cl. Spec. Mstr. Mar. 25, 2025) (Respondent did not preponderantly demonstrate factor unrelated of CMV infection as causal of petitioner's GBS in flu-GBS Table claim).

[30] I have done that very thing in many other cases, and my determinations have been subsequently upheld. For example, I denied entitlement in a case in which a claimant alleged the Tdap vaccine had caused him to experience GBS. *See K.A. v. Sec'y of Health & Hum. Servs.,* No. 16-989V, 2022 WL 20213037 (Fed. Cl. Spec. Mstr. Apr. 18, 2022), *mot. for review den'd,* 164 Fed. Cl. 98 (2022), *aff'd*, 2024 WL 2012526 (Fed. Cir. May 7, 2024). I found (among other things) that the Petitioner's personal medical records suggested that an upper respiratory infection he had experienced around the time of his GBS onset might have been causal of it, and therefore that this fact pattern inhibited his ability to meet the second *Althen* prong. *K.A.,* 2022 WL 20213037, at *30–32. Many special masters have found *Althen* prong

The second prong is not a meaningless obligation, as the Circuit has noted. *Capizzano*, 440 F.3d at 1327. Petitioners do not simply prove the "can cause" element, and then rely on the fact of the injury itself to do all the work in establishing a "logical sequence of cause and effect." Rather, in *any* Vaccine Act claim, many factors arising out of the circumstances in which a vaccination occurred can come into play in contributing to a disease or illness—and they are properly weighed even if the prong one, "can cause" requirement has indisputably been met. Petitioners *must grapple* with the actual record before them, and persuasively deal with factors undermining the conclusion that one or more vaccines caused an injury. This does not mean they must disprove alternative causes.

In this case, there are simply too many confounding factors specific to Petitioner's experiences in the six-to-eight-week post-vaccination period for me to conclude it *more likely than not* that the Tdap and MMR vaccines contributed to her AIH. Nor has she demonstrated other factors consistent with her theory (other than the circular point that she in fact developed AIH post-vaccination) that would corroborate it. The medical record simply does not contain sufficient facts suggesting the vaccines had anything to do with Petitioner's disease. The second *Althen* prong has not been preponderantly established.

C. *Althen Prong Three*

Petitioner's showing on this prong requires demonstrating (a) when her AIH most likely began, and then (b) whether that occurred in a medically-acceptable timeframe, measured from the date of vaccination.

On the former fact question, the record does not permit a clear determination of any kind (making it impossible in turn to identify *whether* the timeframe from vaccination to onset of AIH was medically acceptable). Dr. Gish himself was ambivalent in identifying a specific onset date. He deemed significant Petitioner's LFT findings from her late-October 2018 physical, citing them as clear support for the diagnosis. These findings were obtained 68 days, or more than two months, post-vaccination. This is facially a long timeframe, especially in the context of a record lacking much evidence of symptoms progression, or even an identifiable vaccine reaction.

But arguably this could amount to confusing when an AIH *diagnosis* could be made with when Petitioner's disease course *actually began*—the latter being the more important question for purposes of the third prong. *Carson v. Sec'y of Health & Human Servs.,* 727 F.3d 1365, 1369 (Fed. Cir. 2013) (Vaccine Act limitations period begins to run from the manifestation of the first objectively cognizable symptom, whether or not that symptom is sufficient for diagnosis). It is highly likely Petitioner's AIH began before the late October test results were obtained—and at trial Dr. Gish seemed to embrace an onset earlier than late October.

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one met in such cases—but this determination would not mean the same for prong two, if other factual evidence undermined the vaccine as specifically causal in a petitioner's own circumstances.

Some faint evidence of fatigue and nausea experienced by Petitioner over a several-week period, from early September to mid-October, could constitute her AIH onset. These symptoms are nonspecific for AIH, while also being consistent with other comorbid concerns Petitioner suffered from, such as kidney stones or gastrointestinal issues (like GERD). But they are also reasonably associated with AIH. At the same time, Petitioner never reported any concerning or unusual reaction to the vaccines she received, and there is no record proof (clinical or testing results) of the presence of concerning inflammation in September or October 2018 either. And the purpose of Petitioner's October physical was *not* to address any then-alarming symptoms, nor does it appear that Petitioner reported such concerns at that time (despite Petitioner's contentions about daily nausea that month).

All of the above makes it exceedingly difficult to pinpoint an onset date for evaluation under the third *Althen* prong. Treaters like Drs. Oloruntoba and Wu seem to have assumed, however, that Petitioner's overall course—fatigue and some nausea after return from her travels, then the inadvertent discovery of LFT elevated levels, culminating in her December 2018 presentation—reflected a single disease process. Thus, she experienced some clinical features of early AIH within six to eight weeks of vaccination, later corroborated by the LFT test results, and progressing to more concerning symptoms that led her to seek treatment in December, after which she was more formally diagnosed. Such an onset would fall into the temporal interval proposed under Dr. Gish's theory. Indeed, even Dr. MacGinnitie seemed to agree that an autoimmune process triggered by the immune stimulation of a vaccination might first manifest within six to eight weeks of the trigger. Tr. at 176.

Accordingly—had I found the Tdap and MMR vaccines could cause AIH in the manner proposed by Dr. Gish, I would also be able to find Petitioner's onset occurred in a medically-acceptable, post-vaccination timeframe. Of course, *I have not found* prong one was satisfied—and therefore it does not matter that Petitioner's likely onset is consistent with that theory. I also note that the vagueness of the evidence relating to onset is comparable to the same overall lack of record support for prong two. Just as Petitioner's own medical record does not preponderantly establish vaccination as the likely actual cause of her AIH, that record also makes it exceedingly difficult even to say when her AIH began.

III. Comment on Application of Preponderance Test to Petitioner's Showing

The Court has concurred with my enunciation of the preponderance test as *individually* applicable to each *Althen* prong. Remand Order at 37–38. But the Remand Order nevertheless expressed concern that my vacated entitlement decision inadequately explained why this test was not met overall. *Id.* at 44.

Hopefully the exhaustive re-review and discussion of the record I have now provided assuages those concerns. But it is possible that Petitioner will seek further review of my decision, arguing once again that her evidentiary showing did in fact meet the preponderance test. I will

therefore conclude with a few points about the application of the preponderance standard in Vaccine Act claims.

Preponderance is not met on the basis of the *amount* of evidence filed. 100 items of evidence do not "beat" 50 simply due to numerical superiority, or the physical weight of paper if placed on a scale. And claimants do not "double-prove" their case by offering multiple overlapping items of literature that effectively say the same thing. What matters is the *individual reliability of the items of evidence offered*, coupled with how well each item knits together into an overall theory that does not amount to speculation or unreasonable extrapolation.

Similarly, establishing preponderance does not turn on the *fact* that a claimant has offered something in support of their claim. Determining causation is not an exercise of ensuring that each *Althen* prong "bucket" has been filled with *some* evidence that is not obviously fraudulent or false. Rather, special masters are expressly called to *weigh* the totality of evidence offered, pro and con. In so doing, they make reliability findings for theories based on the chain of propositions or building-blocks offered. And in performing these assessments, they may find that a petitioner's showing fails to cross the "more likely than not" line—even if individual items of evidence still merit weight.

In addition, the fact that vaccine injuries are (thankfully) rare events does not excuse a claimant from the burden of the preponderant test—nor does it mean that preponderance becomes a function of what evidence is available at a given time. As the Court has recognized, "the standard of proof does not operate as a sliding scale that varies depending upon the quantity and quality of the scientific evidence that is available." *Caves v. Sec'y of Health & Hum. Servs*., 100 Fed. Cl. 119, 143 (2011), *aff'd*, 463 F.App'x 932 (Fed. Cir. 2012). Even if there is generally little in the way of independent research or findings bearing on a proposed theory to begin with, petitioners do not get graded on a curve.

My evaluation of this claim also does not amount to a heightening of the preponderant burden, requiring certainty even though the evidentiary test does not. It has been my experience as a special master that I can *never* be certain a vaccine did, or did not, cause a given injury—regardless of the case's outcome. Special masters are called upon only to evaluate the *likelihood* of causality, based on the totality of evidence. Cases turn on how, and in what direction, that evidence tips.

Here, a preponderant evidentiary showing *has not been* made—and it was not a close case. This is yet another instance in which a claimant wants to leverage a set of basic facts about a disease process and immunology into an explanation for pathology. But that is difficult to do—even if the core concepts are reasonable. As a special master, I have in the past 12 years reviewed hundreds of claims. In nearly every such disputed case where the injury was autoimmune in nature, a good faith argument could be made that vaccination (even where distantly-administered in time) *could* set up a process leading to the injury, simply due to the obvious fact that vaccination impacts the human immune system. There are any number of experts prepared to offer an opinion sketching

out the process of how that might occur, relying on what is medically/scientifically known about immunology. And they do so invariably in "a field bereft of complete and direct proof of how vaccines affect the human body," (*Althen,* 418 F.3d at 1280).

Even so, *such claims do not automatically prevail.* To meet the preponderant standard, sufficient evidence must be presented to allow for the conclusion that the vaccine "more likely than not" acted as proposed. That was not accomplished in this case, even though Petitioner has offered some reliable evidence plus an experienced hepatologist to convey her causation opinion.

CONCLUSION

Petitioner has had her day in Court. I was present for all testimony offered therein. As I initially discerned, and as I now reiterate after a second review of the entirety of the record, she did not meet her burden of proof. Accordingly, because this claim has not been supported with sufficient preponderant evidence, Petitioner is not entitled to compensation.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[31]

IT IS SO ORDERED.

s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[31] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.